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CONTENTS

The Association of Diabetes and Tuberculosis, Epidemiology, Pathology, Treatment, and Prognosis. *...ard F. Root.* (To Be Continued.) 1

Sugar Tolerance in the Arthritic. *H. Archibald Nissen* and *K. A. Spencer.* (To Be Continued.) 13

MEDICAL PROGRESS

Cardiovascular Review for 1932. *Paul D. White.* (Concluded.) 20

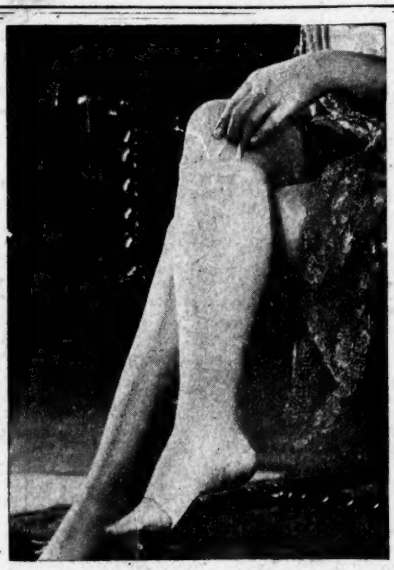
CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Case 20011—*Augustus S. Rose, Arlie V. Bock, Tracy B. Mallory, George W. Holmes* and others. Case 20012—*Augustus S. Rose, William B. Breed, William D. Smith, Tracy. B. Mallory, Wyman Richardson* and *Howard B. Sprague* 36

EDITORIALS

Of What Shall Diabetics Die? 43
Page the Specialist in Fractures! 43
This Week's Issue 44

(Contents continued on next page)



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CONTENTS

(Continued)

MASSACHUSETTS MEDICAL SOCIETY
Postgraduate Instruction 44

BOOK REVIEWS
Migraine. Diagnosis and Treatment. Ray M. Balyeat 58
Obstetrics and Gynecology. Volume II 57

BOOKS RECEIVED FOR REVIEW 58

MISCELLANY
An Artificial Esophagus 47
A Radio Message Prepared and Sponsored by the Committee on Public Education of the Massachusetts Medical Society for the Department of Public Health. Communicable Diseases Among School Children. James A. Keenan 47
Resolutions Adopted at the Twelfth Annual Convention of the American Society of Clinical Pathology 49
Resolutions Respecting an Article by Dr. Morris Fishbein 47
Where the Typhoid Fever Deathrate Is Still High 35

NOTICES 50

RECENT DEATHS 50

REPORTS AND NOTICES OF MEETINGS 51

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES 56

The Massachusetts Medical Society

The next Annual Meeting of the Massachusetts Medical Society will be held in Worcester, at the Hotel Bancroft, Monday, Tuesday and Wednesday, June 4-5-6, 1934.

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THE ASSOCIATION OF DIABETES AND TUBERCULOSIS*

Epidemiology, Pathology, Treatment and Prognosis

BY HOWARD F. ROOT, M.D.†

I. EPIDEMIOLOGY

OUTLINE

1) Introduction
2) Mortality from tuberculosis among diabetics at autopsy
3) Tuberculous infection and morbidity
4) Falling mortality rates from tuberculosis
5) Rising diabetic mortality rate
6) Present-day epidemiology
   a) childhood infection and mortality rate
   b) clinical tuberculosis in childhood
   c) adults
   d) other factors
      (1) Contact
      (2) Occupation
      (3) Age and Sex
      (4) Social and Economic
      (5) Race

1) INTRODUCTION

IN studying 245 cases of associated diabetes and pulmonary tuberculosis the following facts appeared outstanding:

(a) The development of pulmonary tuberculosis in juvenile diabetics occurred more than ten times as frequently as among non-diabetic Massachusetts grade and high school children.

(b) Pulmonary tuberculosis developed in 8 per cent of diabetic patients within three years of recovery from coma.

(c) The incidence of pulmonary tuberculosis in adult diabetics is increasing despite the general decrease of tuberculosis mortality with consequent reduction of contacts in the community.

(d) In 85 per cent the development of progressive tuberculosis appeared to follow the onset of diabetes.

If such a predisposition to the development of pulmonary tuberculosis is induced by diabetes mellitus, an explanation should be sought in the answers to certain hypothetical questions.

(1) Does diabetes itself by reason of its metabolic disturbances so change the tissues as to provide a culture medium especially favorable for the development and propagation of the tubercle bacillus? Or does diabetes lower the resistance and prevent the development of immunity to tuberculosis?

(2) Do diabetic families have inherited predisposition to tuberculosis as well as an inherited diabetic tendency?

(3) In the cases so far reported has there happened to be an exceptionally frequent contact with open tuberculosis in family or friends?

(4) Does diabetes chance to develop in persons whose habits of life, occupation and economic status favor the development of tuberculosis?

All students have agreed that diabetes does not develop in patients with tuberculosis more frequently than in the non-tuberculous population. For a demonstration of the incidence of pulmonary tuberculosis in diabetics one must consider not only varying standards of diagnosis, varying social conditions in communities from which cases are reported, but also more important still the remarkable changes in mortality rates for each disease observed during the last 50 years.

2) MORTALITY FROM TUBERCULOSIS AMONG DIABETICS AT AUTOPSY

During the latter half of the nineteenth century the diabetic patient appeared doomed to die of pulmonary tuberculosis if he succeeded in escaping coma. In 1883 Bouchardat, the great French student of diabetes, stated in his text that at autopsy every case of diabetes had tubercles in the lungs. Earlier in the century Griesinger[1] had found tuberculosis in 42 per cent of 250 diabetics, and Frerichs[2] found in 55 autopsies 21 cases of pulmonary tuberculosis.

Windle[3] is frequently quoted as stating that in over 50 per cent of 333 diabetic autopsies pulmonary tuberculosis was found. His paper, presented as a thesis for the degree of M.D. in the University of Dublin in 1883, consisted of a summary of autopsies obtained largely from the literature and in part from the General Hospital, Birmingham. Accordingly they represent in large part the same cases as were reported in other early articles. At this time Windle had examined only one case for the newly discovered tubercle bacillus and had failed to find it present. In Windle's series two cases showed

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tubercles in the liver, one had tubercles in the spleen, five had tubercles in the kidneys and two had tubercular ulcerations of the intestine. So striking was the association in the minds of the physicians of the time that several writers believed there to be an hereditary tendency to the two. Thus, Croner[4] relates the occurrence of an hereditary and familial tendency to tuberculosis in 37 out of 100 diabetics. Twelve others in this same series had tuberculosis. He quotes Schmidt who believed that there was a diabetic predisposition in tuberculous families. Purdy[5] earlier had noted that the territories furnishing the highest mortality from diabetes in the United States coincided closely with those furnishing the highest mortality from consumption. One is forced to comment, however, that this would be true in the most thickly populated territories where also diagnostic facilities would be best. In Table 1 are summarized representative autopsy reports of active tuberculosis in diabetics and non-diabetics. Naunyn[6] marked an epoch in the history of diabetes, which may also be used as the boundary between the old and the modern in tuberculosis study. Up to Naunyn, 1906, tuberculosis had been reported in 264 out of 622 diabetic autopsies or 42 per cent. In the 25,712 autopsies from 5 general hospital series including Klebs' report in 1909 active tuberculosis was found in 7955 cases, or 30.9 per cent.

These statistics of the frequency of tuberculosis in diabetes were largely based on autopsies performed upon patients under treatment in the large municipal hospitals in European cities. As suggested by Windle, doubt may legitimately be felt regarding the diagnosis in the cases studied before the discovery of the tubercle bacillus and before the general use of methods for its identification in the clinical study of cases. It should be remembered that in the latter part of the 19th century there was little segregation of tuberculous patients from non-tuberculous and that in those hospitals patients remained for long periods of time, and in the case of the poor, even to death.

Striking as these figures are, one should nevertheless compare them with statistics for the general population and particularly for the whole hospital population. The prevalence of tuberculosis was undoubtedly far greater than was suspected.

Klebs[11] in 1909 reviewed ten series of autopsies in children 0 to 15 years of age totalling 5308 cases. Of these, 1247, or 23.5 per cent were tuberculous. These series were published between the years 1889 and 1909. If deaths under one year were omitted, the percentage would be much higher. For example, the percentage would be 44 in Muller's series, 55 for Hamburger and Sluka, and 40 for Sehlbach. A comparison of the percentage of tuberculous cases in diabetic autopsies with a non-diabetic series therefore may lead to gross errors unless all corrections for such factors as age, sex, changing diagnostic standards, and morbidity rates are applied by modern statistical methods.

The difficulties in drawing conclusions are illustrated by comparison with the association of tuberculosis and cancer. Pearl[27] found among autopsied cases of carcinoma at Johns Hopkins less than one-half as many cases with associated active tuberculosis as were found in series exactly comparable in number, age and sexes, but free from carcinoma. His inference that an antagonism exists between the two diseases had been disputed by Carlson and Bell[15] who showed that a similar low incidence of tuberculosis occurs in cases with chronic heart disease. Figure 1 (reprinted from Carlson and Bell) presents the incidence of active (excluding healed) tuberculosis, cancer and heart disease in 11,195

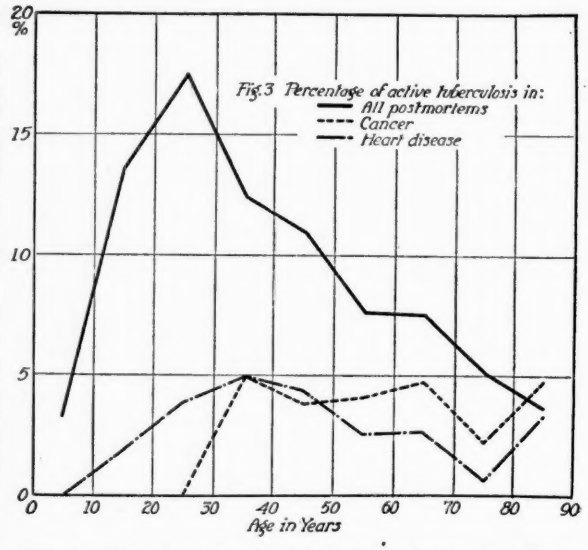

FIG. 1. Frequency of active tuberculosis at autopsy. (Carlson and Bell.)

postmortem protocols. Eleven thousand of the records were from the Department of Pathology of the University of Minnesota, and 195 from the Glen Lake Sanitarium for tuberculous patients near Minneapolis, Minnesota. Although the association of tuberculosis with heart disease is shown to be less than its association with cancer, a more important point for our discussion is emphasized, namely, that the frequency of the association of tuberculosis with either cancer or heart disease is much less than the incidence of tuberculosis in the group as a whole. This group is a heterogeneous one, including many patients who died of tuberculosis alone. This point is made more definite by Wilson and Maher[74], who in computing the probabilities of an association of tuberculosis and cancer, estimated that in Pearl's series the association of the two diseases was really about three times as great as would be expected. Carlson and Bell

Morton in 1694 reported the first case of the combination. John Rollo in 1798 gave the clinical and postmortem records of a case of diabetes evidently complicated by pulmonary tuberculosis with cavity formation.

hold that patients usually die of a single major disease, aside from arteriosclerosis and terminal infections. The proper control for the association of active tuberculosis and any chronic disease is the incidence of active tuberculosis with some other chronic disease, with a similar age incidence, for example, heart disease. Cancer and heart disease, like diabetes, are much less frequent than tuberculosis as causes of death in early life and the association of tuberculosis with these two may therefore be compared with the association of diabetes and tuberculosis. In figure 1 it is apparent that at no age period before 80 years is the association of these two diseases with tuberculosis on the average as much as one-half as frequent as is active tuberculosis in the series as a whole.

The large number of cases in Carlson and Bell's series justifies the conclusion that if the percentage of diabetic patients showing active tuberculosis is about the same as in the general population it is an abnormally frequent combination as compared with the combination of either heart disease or cancer with tuberculosis. Roughly it might be estimated under these conditions as being from two to three times as frequent as expected. Therefore, again referring to table 1, the fact that for the entire series the incidence of active tuberculosis at autopsy in the diabetics is 28.4 per cent and 22.9 per cent for the non-diabetic indicates a frequency of tuberculosis probably two to three times as great in the diabetic as would be expected.

The most important criticism of autopsy statistics depends upon the fact that the duration of diabetes is not given sufficient weight. The average duration of life after onset of diabetes was so short in the older series that no fair opportunity was afforded for the development of tuberculosis. This point comes out quite clearly in our study of tuberculosis in diabetic children and will be alluded to later. In order to form an opinion as to the present incidence it is necessary to consider the changing morbidity and mortality rates for the two diseases as well as modern clinical and pathologic data.

3) Tuberculous Infection and Morbidity (Incidence)

It is impossible to present with any accuracy the incidence of tuberculosis in terms of illness. Since the survey carried out by the Framingham Community Health and Tuberculosis Demonstration in Framingham, Massachusetts, disclosed nine active cases for every death, it has become the custom to rate tuberculosis cases as being ten times the number of deaths in any community. The total number of persons ill with tuberculosis in the United States would therefore be estimated to be about 1,000,000. This estimate may be compared with Dublin and Joslin's estimate of approximately 250,000 to 400,000 individuals in the United States who have diabetes and about 2,000,000 who either have or will develop it.

The incidence of tuberculous infection is indicated by means of skin tests with reasonable accuracy. In addition autopsy reports have been carefully analyzed with regard to the evidences of latent or healed tuberculosis as well as active forms. Classic observations testify to the former universality of tuberculosis in European cities. The records of Naegeli[8] (1900) showed that definite signs of tuberculosis were found in 97 per cent of bodies examined consecutively by him at Zurich, that is, of persons dying in a general hospital from all sorts of diseases and accidents. Reinhart[13] found the remarkable fact that 32 per cent of his cases in Berne presented evidence of progressive lesions. Approximately one-third of all the patients who terminated their days from a variety

TABLE 1

Incidence of Active Tuberculosis at Autopsy

General					*Diabetic Only*				
Author	Period	No. Cases	Tuberculous		Author	Period	No. Cases	Tuberculous	
			No.	Per cent				No.	Per cent
Lubarsch[7]	1888	6536	2688	41	Griesinger[1]	1859	250	105	42.0
Naegeli[8]	1900	508	198	39	Frerichs[2]	1884	55	21	38.2
Zahn[9]	1902	6320	2058	33	Honl[17]	1898	29	12	41.4
Norris[10]	1904	7040	1764	25	Williamson[18]	1898	24	12	50.0
Klebs[11]	1889-1909	5308	1247	23.5	Sauvage[19]	1895	42	7	16.7
Steinmeier[12]	1914	7231	1321	18.2	West[20]	1902	50	24	48.0
Reinhart[13]	1917	360	117	32.0	Seegen[21]	1893	122	63	51.6
Suzue[14]	1927	4207	890	21.1	Naunyn[6]	1906	50	20	40.0
Carlson and Bell[15]	1929	11195	960	9	Montgomery[22]	1912	25	6	24.0
Ophuls[16]	1926	3000	632	22	Golden[23]	1908-1925	53	14	26
					Fitz and Murphy[24]	1924	37	7	19
Total		51705	11875	22.9	Warren[25]	1930	283	13	4.6
					Pagel and Henke[26]	1932	101	15	15
					Total		1121	319	28.4

Norris—Only cases showing tuberculosis of lungs, pleura, pericardium, heart and great vessels counted.
Klebs—Children 0 to 15 years.
Suzue—Cases over 20 years of age.

of causes within a large general hospital were the subjects of advancing tuberculosis, while, of the remaining, 64 per cent presented evidence of healed tuberculosis. More recently, and especially in American communities, long series of postmortem examinations have yielded less evidence of tuberculosis. Ophuls[16] in San Francisco and Carlson and Bell[15] in Minneapolis report much lower percentages. Thus Ophuls (p. 170), comparing his series with 3 large autopsy series, found the incidence of healed or inactive tuberculosis to increase from 14 per cent in the second to 63 per cent in the seventh decade. Much depends upon the criteria adopted for the diagnosis. No very rigid comparison may be made with the incidence of healed tuberculosis at autopsy. The series of Carlson and Bell only shows 13.2 per cent in the eighth decade. Other evidences of decreasing infection in American communities appear in the examinations of school children in Framingham. The per cent of positive reactions to the tuberculin skin test in Massachusetts grade school children fell during 5 years from 28 to 17, according to Lord. On the other hand calcified tracheobronchial lymph nodes and areas of calcification in parenchymal tissue have occurred in approximately 75 per cent of chest roentgenograms of 1659 diabetic patients at the Deaconess Hospital. (See table 5.)

4) Falling Mortality Rates for Tuberculosis

A striking fall in the death rate from tuberculosis in the total population has occurred during the last century. Pearson[28] and others consider that the decline in the death rate from tuberculosis indicates the subsidence of a world epidemic of tuberculosis due to an increase in racial immunity. However, it is true that other infections have similarly declined and it is unlikely that any racial immunity has been developed to all infections at the same time. The fall in death rate has varied widely in different communities in a manner more consistent with economic conditions and with efforts to control the disease than with any general increase in immunity. The death rate from tuberculosis (all forms) in the United States has fallen from 400 in 1866 to 69.1 in 1930 (47 states reported by U. S. Public Health Service[29]). In 1930 the Massachusetts death rate for pulmonary tuberculosis was 56.9 and for other forms 7.3, the lowest on record. A decline in the death rate from tuberculosis has occurred in all parts of the civilized world. Had the 1866 death rates prevailed in 1928 in the United States, there would have occurred in the latter years 458,000 deaths. Instead the number of deaths was reduced by 80 per cent. If 300,000 lives were prolonged every year and one out of every 100 developed diabetes, the number of diabetics would be increased by 3000. Although this may seem fantastic, no one would deny that the falling death rate from tuberculosis has contributed definitely to the increasing duration of life in this country. The control of acute infections and the reduction of infant mortality are equally important, but the fact is that the number of middle-aged and aged individuals in the United States has greatly increased. (See table 2.)

In 1930 the population (122,775,046) contained approximately 1,700,000 more individuals between 70 and 79 years of age than would have been the case if the percentage of 1850 still was maintained. Indeed 17.2 per cent of the population were over 50 years of age in contrast to 8.9 per cent in 1850. Dublin[31] states that the number of aged persons increased by 60 per cent while the total population increased only 39 per cent between 1900 and 1920. In New York City the percentage of the population 65 years of age and over increased by 35.7 per cent between 1900 and 1930. The most frequent age of onset of diabetes is 51 years. It is noteworthy that in New York City according to Drolet and Dorr[32] the peak of the death rate from tuberculosis for males has moved forward from the age group 40-44 in 1901 to the age group 55-60 in 1930. The peak of the death rate for females still remains in the age group 20-24 years.

TABLE 2

Increasing Age of the Population of the United States

Age in years	1850 per cent	1900 per cent	1920 per cent	1930 per cent
Under 5 years	15.1	12.1	10.9	9.3
40-49	8.0	10.1	11.5	12.2
50-59	4.8	6.8	7.9	8.7
60-69	2.6	4.1	4.8	5.4
70-79	1.1	1.9	2.1	2.5

The rate of progress toward control of tuberculosis continues and naturally tends to reduce the number of contacts for the diabetic. Unless diabetes in some way specifically increases the liability to the development of tuberculosis, less

TABLE 3

Deaths from Pulmonary Tuberculosis Compared with Total Deaths in 2650 Fatal Cases of Diabetes

	Total Deaths	Deaths from Pulmonary Tuberculosis	
		Number	Per Cent of Total
1898 - June, 1914	342	16	4.7
1914 - Aug. 7, 1922	805	46	5.7
1922 - Nov. 2, 1931	1503	99	6.6

Note: The above statistics are based upon diabetics first seen between 1898 and Jan. 1, 1929, or up to and including Case No. 7564. All deaths reported in the series up to Nov. 2, 1931 are included.

frequent exposure to active cases in the community should reduce the incidence of pulmonary tuberculosis. If, however, prolongation of the diabetic's life, by modern treatment means a longer period for the diabetic tendency toward the development of tuberculosis to act, then cases will not decrease and may actually increase in frequency. That the latter is the case is indicated in table 4 showing the rising mortality from tuberculosis.

TABLE 4

TOTAL AND DIABETIC MORTALITY IN THE REGISTRATION AREA OF THE UNITED STATES

Year	Population in Registration Area, Per Cent	Deaths from All Causes Rate per 1,000 Population	Deaths from Diabetes	
			Number	Rate per 100,000 Population
1880	17.0	19.8	237	2.8
1890	31.4	19.6	1089	5.5
1900	37.9	17.8	2693	9.3
1910	58.3	15.0	8040	14.9
1920	82.2	13.1	14062	16.1
1925		11.8	17385	16.9
1930	96.2	11.3	22528	19.0
1931	96.3	11.1	24331	20.4
1932				22.0

It is important to remember differences in the age at death of the two diseases. The peak of the tuberculosis death rate was formerly reached in early adult life long before the peak of the diabetic death rate. Even in 1932 among policyholders of the Metropolitan Life Insurance Company the average age at death from tuberculosis was 39.8 years whereas the average age at death from diabetes was 61.2 (personal communication from Mr. Herbert Marks). The fall in death rate from tuberculosis after the age of 45 parallels the percentages of the populations in these age groups. In the United States Census report for 1928 out of 80,285 deaths from pulmonary tuberculosis 81 per cent (65,485) occurred before the age of 45 years. In contrast to such an age distribution one must remember that 60 per cent of all diabetes begins after the age of 40 years and that 51 years is the most frequent age of onset, a period of life never attained by the majority of patients with serious tuberculosis.

This difference in age incidence has been insufficiently stressed in comparisons of the incidence of diabetes in the tuberculous with the incidence of tuberculosis in diabetics, but is clearly shown in Bolduan's[33] analysis of statistics for New York City in table 6.

5) RISING DIABETIC MORTALITY RATE

Coincident with the falling death rate from tuberculosis, the death rate from diabetes has undergone a most extraordinary increase. In fact, there is no other disease about which we have reliable statistics which has made such a startling increase in the United States. In table 4 are shown the total and diabetic mortality in the Registration Area of the United States, according to Joslin, Dublin and Marks[34].

Similar increases in mortality rates are shown by Life Insurance, State and City statistics, especially in the larger cities. Indeed, the fact that the greatest increases are shown in communities where medical and diagnostic facilities are most modern is consistent with the belief that part of the increase is due to more frequent urinalyses, periodic health examinations and insurance examinations. The increasing duration of life allowing hereditary tendencies to become manifest is clearly a most important factor. Possibly the urbanization of the population with less manual labor is an important factor. The community contains relatively more diabetic individuals now than the mortality rates indicate since with modern treatment the duration of life has been so greatly increased. The ages at death of diabetes have changed accordingly. In 1890 the diabetic death rate per 100,000 in the Registration Area of the United States for 45 years and over was 20.6 whereas in 1920 it was 57.3. The change in ages at death of diabetes is better shown by the rates for Massachusetts cited by Joslin. Since 1922, the mortality for those under 50 years has been cut in half and for those over fifty years of age has been doubled. From 1921 to 1925, 86 per cent of all diabetic deaths in Massachusetts took place at or above the age of 50 years[35].

In Toronto, also, whereas in 1918 to 1920 the percentages of deaths from diabetes occurring above and below the ages of 50 years were about equal, since that period a steady advance in age at death has occurred until in 1931, nearly 90 per cent of the diabetic deaths occurred over the age of 50 years. (Joslin et al[36].) The greater longevity of the diabetic increases the opportunity for infection.

A great increase in the number of diabetic deaths in females has occurred. In summary, five facts of fundamental importance have come out. 1) The former almost universal infection of the community with tuberculosis beginning at an early age is diminishing. 2) The prolongation of life of the tuberculous patient is shown by the falling death rate and delay in the peak of the death rate. 3) The great increase in the number of diabetics in the community is due in part to better diagnoses, better treatment and the increased longevity of the population as a whole bringing more people into the diabetic age period. 4) The average duration of life of the diabetic is steadily increasing with consequent increased opportunity for the development of tuberculosis, if exposure to open cases occurs, or if diabetes exerts a specific influence in its development. 5) The mortality from tuberculosis among diabetics has in-

creased in spite of the general reduction in tuberculosis mortality.

6) Present-Day Epidemiology of Associated Diabetes and Tuberculosis

a) *Childhood.* The development of pulmonary tuberculosis in school children has been thoroughly studied so that comparison of its frequency in diabetic children is more reliable than ever before. White[37] estimates the total number of diabetic or pre-diabetic children in the United States as 25,000, basing her analysis on the fact that in 1922, the number of diabetic deaths in childhood was 1,080. This represented 1 in 8000 children in the population. Since the duration of life then was only about one year, the number of deaths would represent the number of new cases per year. Assuming that since the use of insulin no deaths occurred, there should be approximately 10,000 cases, and with an allowance of 1,000 per year for the fifteen years of childhood, a total of 25,000 cases might be credited to diabetic or potentially diabetic children. The frequency of diabetes in childhood is better pictured if we realize that there are as many children who have the disease before they are ten as there are men and women who develop it after they are seventy.

In considering the incidence of tuberculosis in diabetic children, the duration of diabetes is of prime importance. Without observation through the period of adolescence no adequate idea of the probable effect of diabetes upon the development of tuberculosis will be obtained. The series of chest roentgenograms upon 280 children included in table 5 disclosed only one case of chronic pulmonary tuberculosis and one early acute case. These children and adolescents were not acutely ill and the series is not therefore comparable with a general hospital series in which patients would be included who had been sent to the hospital because of illness. However, within three years, repetition of x-rays showed three more of these patients to have developed adult pulmonary tuberculosis.

The evidence of postmortem examination is of doubtful value for the same reason, namely, that few autopsies have been performed upon children with diabetes of prolonged duration. Tuberculosis was not found in 13 autopsies performed upon children with onset of diabetes in infancy (White[37], page 208). In three cases the duration of diabetes was between 1.1 and 2.1 years, in seven the duration was 0.3 years or less, and the duration in the remaining three was unknown.

Warren[38] reports twenty-two autopsies upon diabetic children without pulmonary tuberculosis. Later at the Deaconess Hospital one death from extensive pulmonary tuberculosis occurred in a boy, Case No. 7041, who died at the age of fourteen years after diabetes of eight years' duration. Only two other cases among his twenty-four (2 cases added since publication of the 22 mentioned) had a duration exceeding 1.9 years. In the older literature cases in childhood were not rare. Naunyn reported four. Pagel and Henke[26] (see Chap. II, *New Eng. J. Med.*, Jan. 11, 1934) had only one case with tuberculosis in thirteen diabetic autopsies in the first decade.

The incidence of infection as shown by the Mantoux test and the x-ray evidence of calcified tracheobronchial glands is comparable in frequency with the results of studies carried out in Massachusetts school children by the State Department of Health. Chadwick and Zacks[39] report a study based on 42,071 children from 5 to 15 years of age examined physically and given the Pirquet tuberculin test during a period of three years. The ratio of reactors in the 42,071 children studied in this series shows a gradual trend upward from 21 per cent infections at age five, and 28 per cent at age ten, to 35 per cent at age fifteen. In 1929-1930 among 7,318 high school students tested in these clinics 39 per cent reacted, according to Pope, quoted by Lord[40]. The average percentage for the entire group of 275,252 children including the school year 1930-1932 is 25, according to Lord[41]. Hetherington, Opie and their associates[42] also found that by age the ratio of reactors rises consistently from about 15 per cent at five years to 50 per cent at the end of high school, using 0.01 mgm. of old tuberculin intracutaneously. Different sections in the city may show a wide variation in the percentage of reactors. It was found in one city of 60,000 that the number of children reacting to the tuberculin test in different school districts varied from 11 to 60 per cent. The number of reactors increases with the opportunities that the child has for contact with bacillus carriers. Persons with chronic forms of pulmonary tuberculosis, such as are found in granite quarry workers, expose and infect more children than persons with more acute forms, who live a much shorter time after the disease becomes infectious. Crowded living conditions afford more opportunities for contact and congested areas show a higher percentage of infection. Twice as many reactors are found among children with a history of direct contact with a case of pulmonary tuberculosis. Another factor to be taken into account is bovine infection. In a few rural towns with a scattered population and less than the average number of pulmonary tuberculosis deaths, 38 per cent of reactors were found. The majority of the small towns, however, show a little less than the average percentage for the state as a whole. The boys of the Irish, Canadian and Teutonic groups in this series show a much higher incidence of infection than the girls. The death rate in Massachusetts for all forms of tuberculosis declined from 144 per hundred thousand in 1917 to 73 in 1927.

The scarification method rather than the Mantoux test was used in the Massachusetts

studies. Although the percentage of reactions was accordingly somewhat reduced, according to Pope, it is unlikely that cases of active pulmonary tuberculosis were missed. Dickey and Seitz[43] in San Francisco used the intracutaneous method upon 3600 children and found 6.8 per cent reactions at the age of 14 years.

In diabetic children out of 97 tests by the Mantoux method 46 per cent have been positive. In Table 8 (Chap. III, *New Eng. J. Med.* Jan. 18, 1934) these cases are listed by age. It is apparent that most of them are in the older groups so that the incidence is comparable with the Chadwick data. It should be noted however, that the Chadwick data were based on the scarification method of von Pirquet which gives consistently fewer positive reactions than the intradermal method of Mantoux. Our children received in nearly all cases 0.1 cc. of 1:1000 dilution of old tuberculin. In certain cases failing to react with this dose 0.1 cc. of dilutions of 1:500 or 1:100 were used. Naturally a higher percentage of reactions are obtained if multiple tests are used, as illustrated by the results of Doolittle[44] who obtained 75 per cent reactions with intracutaneous tests carried to 1 mg. as compared with 37 per cent by the scarification method alone. When these facts are considered, one might conclude that the apparently greater incidence of infection among diabetic children so far tested may be more apparent than real, especially as the small number tested does not allow a fair comparison. However, a consideration of the roentgenologic and clinical data confirms this greater incidence of tuberculosis.

The great frequency of tuberculous infection in diabetes during childhood and adolescence appears in a consecutive series of roentgenograms of the chest carried out by Dr. L. B. Morrison and Dr. I. K. Bogan, and summarized in table 5. These patients had been admitted to the Deaconess Hospital for treatment of diabetes and for the teaching of parents. They were not acutely ill. The classification "tracheobronchial adenitis" included visible and slightly enlarged glands at the hilum. The group in which definite calcification of tracheobronchial glands occurred is the largest and may be assumed to represent healed or partially healed tuberculous infection. Thus, 42 per cent of the cases in the first decade and 70 per cent in the second decade presented this finding. Suspicious cases showed slight increase in markings usually toward the apex. From this group have come four cases of pulmonary tuberculosis within three years. In summary it appears that tuberculous infection in diabetic children is definitely of greater frequency than among school children of Massachusetts, Philadelphia or San Francisco.

b) *Clinical tuberculosis in childhood diabetes.* Among 750 diabetics with onset of the disease before fifteen years of age twelve have subsequently developed pulmonary tuberculosis of the adult type. In eight cases there was a definite history of contact with tuberculous cases. Six were girls and six boys. In addition tuberculosis of the skin has occurred twice, tuberculous peritonitis once and broken-down cervical adenitis once.

In only three of the twelve cases did tuberculosis develop before the age of fifteen years, in the remainder tuberculosis was not recognized for periods of 2.5 to 8.0 years after onset of diabetes. Consequently the rate of incidence must be compared not with the incidence at first examination of school children but with incidence after a follow-up of three years. Fortunately data from the Chadwick clinics are available for Massachusetts. Pope[45] reports on the results of a follow-up of 5000 school children from two to six years after they were classified as having pulmonary tuberculosis, childhood tuberculosis or as suspects. Thirty new cases of pulmonary tuberculosis developed in this group, nine in boys and 21 in girls. The average age of diagnosis was 15.4 years as compared with 16.7 years for our twelve diabetics. The proportion of cases developed in the childhood type group (with visible or calcified tracheobronchial glands) was twice as high as among the suspect group.

In this observation group of 5000 cases then, pulmonary tuberculosis developed in 0.6 per cent. The incidence of pulmonary disease in the total school population was 0.1 per cent. From the total number of children, 140,000, it is possible, by adding the number who had pulmonary tuberculosis at first examination and the number who developed it during observation from two to six years afterward, to obtain an estimate with which to compare the diabetic incidence. The incidence in the school population so obtained is 0.12 per cent. The incidence among the 750 diabetic children is 1.6 per cent.

An incidence 13 times that of normal school children is surprising and possibly would not hold in a larger group of diabetics. The proportion is not so outstanding if comparison is made with the results obtained by Hetherington, McPhedran and associates[42] in a study of 4000 school children in Philadelphia. They had 22 cases of pulmonary tuberculosis, or an incidence of approximately 0.5 per cent. Possibly the greater number of colored children in Philadelphia may have influenced their results.

If the adolescent group with onset of diabetes from 15.1 to 19.9 years are studied, the development of tuberculosis is still conspicuously high. From 1898 to 1931, 251 such patients are to be found in Joslin's series. Of this number four have died and four are alive with pulmonary tuberculosis, an incidence of 3.2 per cent. In Massachusetts among 7318 high school children evidence of pulmonary tuberculosis was demonstrated in but 13, an incidence of 0.2 per cent. The incidence of pulmonary tuberculosis is probably underestimated in the diabetic series.

Although our rate of incidence is computed on the entire group of 750 children and 251 adolescents, only a fraction of this group were subjected either to skin test or to chest x-ray. Had the entire group been as completely tested as the school children, undoubtedly other cases would have been discovered. When one recalls that, according to the autopsy data of Carlson and Bell, the combination of tuberculosis with diabetes should be much less frequent than its incidence in a heterogeneous group, the frequency of tuberculosis in the juvenile diabetic becomes relatively far greater.

c) *Adults.* (1) Autopsy Statistics. The frequency of active tuberculosis as found at autopsy varies in different communities and also, with diagnostic standards of the time and of the individual pathologist. Probably there would be little disagreement with regard to the finding of active pulmonary tuberculosis. Apparently tuberculous infection is somewhat less frequent in American general hospitals than in European, as is well-shown in Ophuls' analysis of 3000 autopsies performed in San Francisco in the Hospital of Stanford University. In table 1 only active tuberculosis is included and for present discussion a comparison of statistics should be limited to the period since 1920. In the series of Suzue, Ophuls and Carlson and Bell a total of 18,402 autopsies showed active tuberculosis in 2482, an incidence of 13 per cent. The four diabetic series of Golden, Fitz and Murphy, Warren, and Pagel and Henke yield 49 cases of active tuberculosis in 474 autopsies, or only 10 per cent. As Carlson and Bell have shown, such an incidence of tuberculosis with diabetes is at least twice as great as the incidence of tuberculosis occurring in association with such chronic diseases as cancer or heart disease.

Certain criticisms may be levelled at any comparison of autopsies, and upon analysis it will appear that the comparison made above probably errs on the side of minimizing the frequency of tuberculosis in diabetes. First, the large general hospital series include relatively many more children than the diabetic series. Thus, in the non-diabetic series of Carlson and Bell, 25 per cent of the total were less than ten years of age, whereas in Warren's diabetic series only 3 per cent fell in this age period. Over 15 per cent of all cases of tuberculosis in the Carlson and Bell series occurred in cases under twenty years of age. In Ophuls' series 9 per cent of all active cases of tuberculosis occurred under the age of ten years and 12 per cent of the 3000 cases autopsied were under twenty years of age. The high incidence of tuberculosis as a cause of death in young children overloads general hospital statistics in comparison with a diabetic series.

Secondly, the factor of prolonged duration of diabetes is not properly presented in the autopsy series. Among the 245 clinical cases of this series, 49 had had diabetes more than ten years. The average duration of diabetes in the active tuberculous cases at the Deaconess Hospital was nine years. Warren[46] collected twenty-three autopsied cases of diabetes with duration of fifteen years or more to which I add two, Case 177 with active pulmonary tuberculosis, and Case 1257 with healed apical lesions. The incidence therefore in the small group is 8 per cent. In table 1 (Chap. II, *New Eng. J. Med.*, Jan. 11, 1934) the average duration of twenty-five cases at autopsy was but 2.5 years and in eight additional cases the age at death is given in the same year as the onset of diabetes. No information is available as to duration in the others. Similarly with most autopsy series upon diabetics, deaths have occurred from coma or sepsis so frequently within one to three years after onset in the past that no fair presentation is afforded of the incidence of tuberculosis under conditions in which an adequate period of exposure to diabetes has elapsed.

TABLE 5

SUMMARY OF 1659 X-RAYS OF DIABETIC LUNGS

	Number Cases
1. No abnormality	187
2. Tracheobronchial adenitis	85
3. Calcification in tracheobronchial glands	1289
4. Doubtful or border-line pulmonary tuberculosis	58
5. Chronic pulmonary tuberculosis	39
6. Acute pulmonary tuberculosis	1
7. Old healed tuberculosis	21

NOTE: The 21 cases of old healed tuberculosis are included in group 3 because all had calcified tracheobronchial glands.

Thirdly, autopsy series in the past have not reflected the changing social and economic status of the diabetic. Not only does he live longer, but because of the beneficial effects of insulin upon his general condition, he lives no longer a life of semi-invalidism and isolation. Mingling at work with the general population in the community means more frequent contacts with open cases of tuberculosis, and more frequent reinfections.

(2) Clinical. The incidence of tuberculosis in diabetics has been studied at the New England Deaconess Hospital by making routine x-ray examinations of the lungs of 1659 cases consecutively, in addition to the usual physical examinations. The results have been divided according to the x-ray findings and are shown in table 5. Classification of the roentgenograms was made as follows:

1. No abnormality.
2. Tracheobronchial adenitis. Visible glands without calcification.
3. Tracheobronchial adenitis with calcification.

4. Doubtful. Slight increase in markings without pulmonary infiltration.
5. Chronic pulmonary tuberculosis.
6. Acute pulmonary tuberculosis. Areas of soft hazy infiltration without scarring or calcification.
7. Old healed pulmonary tuberculosis. Only cases with well-marked fibrous scarring and calcification are included. Less definite and border-line cases have been classified under heading 3, since they always had calcified glands as well.

In this series forty cases of pulmonary tuberculosis were found, two in childhood and thirty-eight among 1373 adults, or an incidence of 2.8 per cent for adults.

Again it should be noted that this group of 1659 cases consisted of patients admitted to the hospital for dietary treatment and instruction, but not for incapacitating or febrile disease. They were not bed patients, with few exceptions. Therefore these results should be compared with the results of community surveys, Draft Board examinations rather than with series of examinations of patients admitted to general hospital wards because of symptoms requiring study or bed treatment. These considerations may have some bearing on the fact that Sosman[47] reported seventeen cases, an incidence of 9 per cent, active pulmonary tuberculosis in a series of 182 consecutive routine chest films in diabetics from the Peter Bent Brigham Hospital. The usual estimate at present is that one per cent of the population has active tuberculosis. Our diabetics therefore had active pulmonary tuberculosis almost three times as often.

(3) Vital Statistics. The statistical evidence of the association may be obtained from clinical studies or from public offices of health. The series here reported of 245 cases from 10,000 cases of diabetes in the series of Dr. E. P. Joslin indicated an incidence of less than 2.5 per cent. The total number of deaths from all causes from 1895 to November 2, 1931 was 2650 of whom 161 representing 6.0 per cent, died of tuberculosis, either pulmonary or miliary. Labbé reports 10 per cent of diabetics in Paris in private practice with tuberculosis in contrast to 20 per cent in public hospitals. During 1930, among 450 diabetics treated at the Hôpital de la Pitie, Labbé[48] reported that among 37 deaths 24.3 per cent were due to pulmonary tuberculosis. Fitz[49] analyzed 1529 diabetic cases admitted to the Peter Bent Brigham Hospital from October 1, 1922 until January 1, 1929 and found 35 who had pulmonary tuberculosis. He estimated that there were 16,000 diabetics with pulmonary tuberculosis in the United States.

Kuelz[50] found pulmonary tuberculosis in only 5 per cent of 692 diabetics. Curschmann[51] found tuberculosis in 4 per cent of his diabetics as also did Rosenberg and Wolf[52] in 1000 cases from Berlin. Delijannis and Petassis[53], in describing 70 cases in Vienna, state that between 1925 and 1931 seven per cent of all diabetic admissions to the hospital had or developed pulmonary tuberculosis. John[54] reported that tuberculosis caused 9.1 per cent of 131 deaths in his 2000 cases. Lyon[55] reported from England in 1930 among 150 diabetic deaths only 11 or 7.3 per cent due to tuberculosis. Murphy and Moxon[56] whose 827 cases of diabetes were chiefly from the rural population of Wisconsin found only 40 cases of tuberculosis, an incidence of 4.8 per cent. Sevringhaus[57] had only eight cases of tuberculosis in 500 diabetics chiefly from the urban population of Wisconsin. Wendt and Peck[58] reported 43 cases among 1073 diabetics in and about Detroit, Michigan. Kramer and Lawson[59] studied the roentgenograms of 408 diabetics in Providence, R. I., and found but five cases, an incidence of 1.2 per cent. The value of such statistics depends upon the duration of the diabetes in the patients as well as the sex, severity of diabetes and frequency of coma. Later examination of this group will be of great interest. Nearly all series of diabetics studied clinically demonstrate an incidence of pulmonary tuberculosis far in excess of the 1 per cent usually accepted for the general population of the United States.

The infrequency of diabetes in tuberculosis sanatoria has been emphasized. Tompkins[60] reported only fourteen diabetics among 4,500 admissions with tuberculosis to the United States Veterans' Hospital at Oteen. Wassmud[61] in 8000 sanatorium cases of tuberculosis in Germany found 60 diabetics. Leitner[62] described in 1930 twelve cases of diabetes among 3500 tuberculous patients in a sanatorium at Bad Reiboldsgrün.

Banyai[63] cites the following statistics: Among 5,224 tuberculous patients admitted to the Muirdale sanatorium since January 1, 1923, there were 31, or 0.59 per cent, who were found to be diabetic. In 1929, Landis, Funk and Montgomery[64], on the basis of 31,834 sanatorium cases treated for pulmonary tuberculosis in 29 sanatoria assumed that the coexistence of these two diseases was between one-sixth and one-third of one per cent. Wassmud in 1927 found diabetes in 60 out of 8,000 tuberculous patients. This amounts to 0.75 per cent.

So far as the incidence of pulmonary tuberculosis in diabetics is concerned, Banyai[63] gives statistics from the recent American literature. In a group of 8,520 diabetics the incidence of pulmonary tuberculosis was approximately 2.6 per cent. His calculations would indicate that tuberculosis occurs about three times more frequently in diabetics than diabetes occurs in the tuberculous. Such a comparison has little meaning unless the ages of the groups are considered. Tuberculous cases in sanatoria are often young. One might as well compare the frequency of diabetes in cases of poliomyelitis.

This is strikingly suggested by the fact that

in 1931 at the Trudeau[65] Sanatorium out of 284 patients listed, only 19 were between 40 and 49 years of age and but one was over 60. There were 169 cases between 20 and 29 years of age. Such a predominance of youth would not necessarily be found in State Hospitals where a selection of young and early cases could not be made. However, a large proportion of the cases with the most serious tuberculosis die before reaching the age when the liability to diabetes becomes greatest. Statistics from tuberculosis sanatoria in the future will probably demonstrate a higher frequency of diabetics. At present there are more diabetics in tuberculous sanatoria who have been referred after treatment for diabetes at the Deaconess Hospital than ever before.

The errors in interpretation which may arise from a disregard of the differing ages at death of tuberculous and diabetic patients are well illustrated by a study of the mortality statistics for New York City by Bolduan in the Weekly Bulletin of the Department of Health[33]. In 1931, among a total of 77,418 deaths, 1,921 were charged to diabetes, making diabetes in a general way responsible for a mortality of 2.4 per cent. However, among 4370 deaths from pulmonary tuberculosis recorded in New York City in the same year only 67 had diabetes entered as a contributory cause or 1.5 per cent. Diabetes thus appears less frequently than would be expected. A similar result is obtained in Massachusetts. Dr. A. S. Pope has informed me that the diabetic deaths form 1.7 per cent of all deaths in the State for the year 1930. Yet among the 19,763 deaths from tuberculosis between 1924 and 1929, diabetes occurred in only 0.9 per cent. In table 6 are shown the relative proportions of total deaths from diabetes and tuberculosis in different periods of life.

When more than 90 per cent of the diabetic deaths occur after the age of 45 years, it is apparent that the above rate of 1.5 per cent for the occurrence of diabetes as a complication of tuberculosis means that tuberculosis does not encourage the development of diabetes. In general, patients with pulmonary tuberculosis who die before the age of 45 years do not develop any more diabetes than the general population. This confirms the older idea that the association of tuberculosis and diabetes is one-sided, i.e., diabetics tend to develop tuberculosis, but tuberculous patients seldom develop diabetes.

The effect of diabetes upon the incidence of tuberculosis must be studied in the age group over 45 years where diabetes is most common. Bolduan has analyzed the tuberculosis register of New York City, containing 18,208 cases. He finds 3239 males and 964 females over 45 years of age. According to the census enumeration, the population of the group over 45 years of age in 1930 was males 732,128, females, 732,049. The incidence of tuberculosis in the two groups was expressed as rates of 4.4 per 1000 males and 1.3 per 1000 females. The average number of deaths of diabetics in persons 45 years of age and over during the two years 1930 and 1931 was males 584, females, 1170. On the basis of the tuberculosis incidence just calculated we might expect 2.5 cases of pulmonary tuberculosis among the 584 males and 1.5 cases among the 1170 females. As a matter of fact, however, the average incidence of pulmonary tuberculosis among the diabetes deaths, in persons 45 years and over during the two years 1930 and 1931, was among males 18.5 and females 23.

Bolduan points out that the registry of pulmonary tuberculosis includes only names of patients whose address is known. Some patients reported cannot be found. The register is not complete. However, there is no reason to believe that the number of actual cases is twice the number in the registry. Even if the calculations were made on the assumption that the register was only 50 per cent accurate, the incidence of tuberculosis in the diabetics over 45 years of age would be 18.5 cases among males, calculated 5; and 23 cases among females, calculated 3.

(d) *Other epidemiologic factors.* For tuberculosis in general in civilized countries no factors are more clearly brought out than the effects of wages, housing, food, occupation and habits. Strikingly low mortality rates are reported in rural states as compared with an industrial and commercial state like Massachusetts. All our 245 cases were from New England, although different racial stocks, occupations, and social habits naturally occurred. City and town dwellers formed 66 per cent. Poverty was a possible factor in only 10 per cent. Alcohol can hardly be discussed. Although in 76 cases there was a record of the use of alcohol, we do not have reliable data as to amounts. Certainly few stigmata of alcoholism were noted. An analysis of racial stocks does not give much help. One hundred and sixty-three were recorded as American, 22 as Hebrews, 12 as Irish, 12 as Canadian and 7 as Italian. Other Eu-

TABLE 6

MORTALITY FROM DIABETES AND PULMONARY TUBERCULOSIS IN NEW YORK CITY

Year	Deaths from Diabetes before the age of 45 years				Deaths from Pulmonary Tuberculosis after the age of 50 years			
	Male		*Female*		*Male*		*Female*	
	No.	Per Cent of Total	No.	Per Cent of Total	No.	Per Cent of Total	No.	Per Cent of Total
1930	62	10.9	77	6.6	841	26.8	283	15.0

ropean nationalities were represented by but a single case.

(1) Contact with active cases. A history of familial tuberculosis or contact in the daily occupation was obtained in 65 cases, with 40 others in which rather indefinite contact may have occurred. Unfortunately we have no accurate data as to the frequency of positive sputum cases among the persons to whom our cases were exposed. Nor can we in most cases form an opinion as to the length of exposure. As a whole the group does not appear to have had unusually close contact with active cases.

(2) Occupation. An indoor or sedentary occupation was recorded in 194 cases or 79 per cent, as compared with 74 per cent in Fitz' series. The occupations with exposure to hard metal or rocky dust are notably most dangerous. Farming is the most favorable occupation so far as the development of tuberculosis is concerned. However, diabetes has no tendency toward the selection of persons occupied in any one trade and in the analyses of 245 cases in this series so great a variety of occupations is found that no single group is very large although sedentary and indoor occupations appear to predominate. Certain cases stand out: as for example, two Jewish butchers who had long been engaged in an occupation where they might have been frequently exposed to tuberculosis while slaughtering animals. Case No. 10548 was a granite cutter and had silicosis moderately far advanced. In Naunyn's 17 adult cases, the occupations were tailor 4, shoemaker 2, office-worker or merchant 3, weaver 1, waiter 1, hairdresser 1, designer 1, laborer 2, mason 1, machinist 1.

The danger of infection during the professional training of nurses and physicians has been repeatedly stressed. Opie[66] and associates have reported the increasing incidence of grave tuberculous pulmonary lesions in medical students with each year of the course. Myers[67] emphasizes the increase in the number of positive tuberculin tests among the probationers in schools of nursing after they have taken tuberculosis services. Cooper[68] finds that the percentage of officers in the medical and dental corps of the United States regular army suffering from tuberculosis is much higher than among other corps. Three out of 50 deaths among diabetic doctors have been due to pulmonary tuberculosis. Two out of five diabetic girls who undertook nurse's training have developed pulmonary tuberculosis and are now in sanatoria. Two other diabetic graduate nurses are known to have tuberculosis.

(3) Age and sex. Age and sex factors are unusual in that almost no deaths from tuberculosis occur in diabetic children. In the past, diabetic children have died too soon for the development of tuberculosis. Among 162 fatal cases of this series no death occurred in the first decade and two in the second. In these two decades about 10 per cent of all cases of diabetes have their onset. Only 27 per cent of the 162 deaths occurred under 40 years. The peak of the mortality curve is in the sixth decade for males and in the seventh decade for females. The usual predominance of the female rate over the male from the tenth to the twenty-fifth year is not shown in the diabetic group. The total number of male deaths is 119, and female 43. The sex distribution for diabetes is about even so that the sex difference in cases developing tuberculosis requires explanation. Possibly the fact that women with diabetes have not in the past undergone pregnancy may be important. Further, diabetic women have been less likely to continue at industrial employment than diabetic men.

Tuberculosis is emphasized as the menace of youth and especially of the period from 20 to 40 years. In later years of life, tuberculosis may be even more serious in its effects so far as the transmission of the disease to other persons is concerned. Cornet[69] stressed the fact that tuberculosis is really a greater danger in old age than in youth if one takes into account the relative number of persons living in the different age levels.

So in diabetes the bulk of active pulmonary tuberculosis occurs late in life partly because 60 per cent of diabetic cases begin after the age of 40, and partly because tuberculosis latent in early adult life becomes more active at that period, either through reinfections or because of the debilitating effect of the diabetes. Among the 245 cases are found 49 cases of pulmonary tuberculosis developing in diabetics after the age of 60 years. Rubin[70] also in analyzing 414 cases of pulmonary tuberculosis past 50 years of age, found that 72 (17 per cent) were diabetics. One must consider loss of resistance due to advancing years as well as due to the development of diabetes. Climate and altitude have not been shown to have any unusual influence in diabetic tuberculosis. In so far as a cold wet climate prevents outdoor life it may have an influence in treatment.

(4) Social and economic factors. Poverty with consequent crowding either in the city or country but chiefly in urban populations has always been a prominent factor. Labbé states that whereas in public hospital wards 20 per cent of diabetic patients are tuberculous, among his private patients the incidence was less than 10 per cent.

Diabetic patients as a group do not belong predominantly to any one social group. The effect of poverty and crowding are demonstrable in selected groups, but there is probably no more diabetes among poor people than among wealthy.

(5) Race. There can be no doubt that certain races do have a lower mortality rate for

tuberculosis than others. Up to July, 1926, Joslin reported only four Jewish deaths from tuberculosis among 143 fatal cases of diabetes in Jews. In the series of 245 cases, 22 were Jews or 9 per cent, whereas Jews formed 13 per cent of 4262 diabetics in Joslin's series. All standards have agreed that the Italian and Hebrew races have rather low tuberculosis mortality, whereas the Negro mortality rate in the United States is more than three times greater than that of the white race, and in urban populations is as much as four times higher. However, local conditions affect these racial rates enormously. Dublin[71] has pointed out that the Hebrew rate is lower in the Bronx than it is in congested portions of lower New York City. So with the Negro rate which is much higher in the city than in the country. Opie[72] has studied the epidemiology of tuberculosis among Negroes. In 1926 the mortality rates in the United States Registration Area for Negroes was 233.2 per cent in the general population, 300.6 per cent in the urban, and 197.4 per cent in the rural population. He questions whether this great incidence is due to (1) inherited susceptibility, (2) the effect of social and environmental condition, or (3) a peculiar susceptibility in adult life because of their failure to develop tuberculosis and some degree of immunity in childhood. Tuberculosis in the Negro is of interest in this country because until recently diabetes was supposedly rare in the Negro race. However, Leopold[73] reported the mortality rate of Negroes from diabetes in Baltimore to be slightly higher than among the white population.

Summary

1) The incidence of active tuberculosis in 1121 diabetic autopsies was 28.4 per cent and in 51,705 non-diabetic autopsies 22.9 per cent. Since the association of the two diseases would be expected less than half as frequently as the incidence of tuberculosis alone, it follows that active tuberculosis occurred in diabetics at autopsy between two and three times as frequently as expected.

2) Tuberculous infection in diabetic children as shown by skin tests and calcified tracheobronchial glands is more common than in Massachusetts school children and the development of adult type pulmonary tuberculosis in a group of 750 children who developed diabetes before the age of 15 years, was more than 13 times as frequent as among Massachusetts school children.

3) Among adolescent diabetics, who developed the disease between the ages of 15.1 and 19.9 years, pulmonary tuberculosis occurred 16 times as frequently as among Massachusetts high school students.

4) Among 1373 diabetic adults, consecutively examined by x-ray, active pulmonary tuberculosis was found in 38 cases, or 2.8 per cent.

5) Deaths from pulmonary tuberculosis among diabetics increased from 4.7 per cent of 342 deaths before June, 1919 to 6.7 per cent of 1503 deaths between August 7, 1922 and November 2, 1931 in spite of the decreasing tuberculosis mortality in the community.

6) The factors of familial contact, race, occupation, housing, poverty, and alcoholism do not appear to explain the greatly increased incidence of pulmonary tuberculosis in diabetes.

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SUGAR TOLERANCE IN THE ARTHRITIC*

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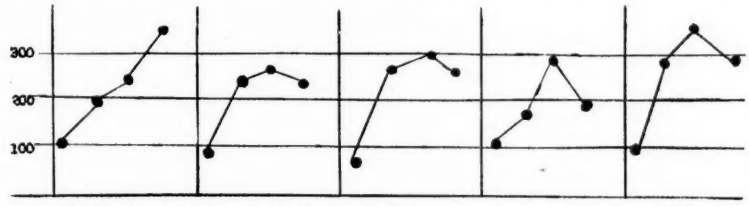
THE problem of arthritis seems to the authors to be this: Why do patients develop joint and general body tissue changes, which in one group progress steadily to marked and final disability, and in another group fail to show the same destruction and disability? The first group is recognized by those men interested in arthritis as the group which has failed to respond to all known forms of treatment. May the answer to the question "What is the etiology of arthritis?" lie in this group?

One is impressed by the multiplicity of measurable systemic changes occurring over a period of years in a group of arthritics. This observation suggests separate, detailed studies of the different body systems and their functions in arthritics.

This paper presents one such study, which deals with the sugar tolerance reaction in 222 arthritics†. Naturally in a study of sugar tolerance reactions in arthritis, a number of vital factors illustrating many different aspects of arthritis are incorporated in order to ensure the validity of the clinical conclusions drawn from these tests. A comparison of the functional status of one group of patients observed over a period of three years or longer was made to determine what prognostic value may be obtained from successive, or single, sugar tolerance tests.

The results of the tests were divided into four groups. The first group showed a normal curve. Normal was one in which the fasting blood did not exceed 120 mg. of sugar per 100 mg. of blood; in which none of the readings exceeded 170 mg., and in which the fourth reading was less than 130 mg. Ninety-five of the 222 arthritics came into this normal group. The second group was termed "Slightly Abnormal." It included those whose curve followed normal lines, but in which one or more of the bloods during the test exceeded 170 mg. In this group there were 51 patients. The third group, termed "Moderately Abnormal," included those whose final blood reading was above 130 mg. of sugar. Thirty-nine patients came into this group. The fourth group, "Markedly Abnormal," showed a tolerance definitely low, the bloods well above 170 mg., and the final blood above 130 mg. There were 37 patients in this group.



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Typical curves found in the Markedly Abnormal Group.

There were but four arthritics among the 222 tested who had a high fasting blood sugar, or who could be considered as diabetics. The remaining patients were not diabetics, and to date have not become diabetics.

A brief explanation of the headings used follows. *Activity* is an arbitrary term, used to designate arthritic activity. Increasing joint involvement, or joint involvement showing itself in local tenderness, pain, heat or swelling (redness of the joints present or not present) was considered the index of activity. *Clinical Heart* is used to describe definite variation from the accepted normal heart function. Under *Nephrop-*

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†Foline method Technique: 1.5 grams dextrose per each kilogram of body weight. Venous blood drawn and sugar content determined. Fasting blood, and three subsequent bloods drawn at three quarters of an hour, an hour and a half, and two and a half hours after the dextrose is ingested.

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athies are those showing variations in urine, not accepted as normal, that is showing intermittently or constantly albumin, casts, red or white blood cells. Under *Sugar in Urine* are those showing green color reaction obtained with the Benedict test if reported constantly for some time, and those showing in addition varying amounts of yellow precipitate, or a measurable quantity of sugar—Under *Tissue Change* are listed those obese, and those showing marked loss of weight,—15 to 25 pounds within a year, and those markedly emaciated and below the average weight standards. *Type 1 Arthritis* refers to the so-called Atrophic or Rheumatoid types. *Type 2,* to the Hypertrophic or Osteoarthritis. *Mixed Type* to those showing a combination of Type 1 and Type 2, plus. To save incessant repetition N.S.T. is used as an abbreviation of *Normal Sugar Tolerance,* and A.S.T. for *Abnormal Sugar Tolerance.*

One hundred and twenty-seven, or 57 per cent, of the total group showed A.S.T. Ninety-five, or 43 per cent, showed N.S.T. Comparison of the N.S.T. and A.S.T. by types of arthritis shows:

	Type 1	Type 2	Mixed Type	Struempell-Marie	G. C. Arth.	Rheumatic Syndrome	Tubercular Arthritis
N.S.T.	44%	20%	12%	15%	5%	3%	1%
A.S.T.	32%	33%	20%	9%	5%	1%	—

Ninety-nine, or 45 per cent of the entire group, were males; 123, or 55 per cent, females. Of the N.S.T. group, 54 per cent were males; 46 per cent females. Of the A.S.T. group, 38 per cent were males; 62 per cent females.

AGE VARIATIONS: In the group with N.S.T. the highest percentage, 36 per cent, were under 30 years: in the A.S.T. group, the highest percentage, 28 per cent, were in the 31-40 decade.

DURATION OF ARTHRITIS: In the N.S.T. group, the highest percentage, 24 per cent, duration 6-10 years; next highest percentage, 21 per cent, duration of months. In the A.S.T. group, highest percentage, 21 per cent, duration 6-10 years; next highest percentage, 13.5 per cent duration of months. This suggests that the actual duration of the arthritis, considered separately, has no marked bearing on the sugar tolerance variations.

FOCAL INFECTION: Tonsils, teeth and antra are said to represent the origin of 85 per cent of infections entering the body. The N.S.T. group showed 59 per cent with tonsils present; 12 per cent with infected teeth; and 6 per cent with infected antra. The A.S.T. group showed 55 per cent with tonsils; 14 per cent with infected teeth; and 6 per cent with infected antra.

This illustrates one of the precautionary warnings suggested in the beginning of the study, that is that no one factor in arthritis can be considered as complete, nor conclusions be drawn from it alone; because if one considered the above figures separately, the natural conclusion would be that the presence of focal infections was nearly a fifty-fifty proposition*.

*Focal Infection in the Arthritic will be taken up in a future paper.

TISSUE CHANGE	Obese	Loss Weight
N.S.T.	3%	35%
A.S.T.	5%	41%

	ACTIVITY	Fever	Elevated Blood Pressure	Clinical Heart	Radiographic Sclerosis	Sugar Urine	Nephropathies
N.S.T.	41%	25%	11%	22%	45%	1%	16%
A.S.T.	48%	41%	16%	31%	36%	17%	23%

ENTIRE GROUP STUDIED—222 ARTHRITICS

TYPES OF ARTHRITIS

Type 1	Type 2	Mixed Type	Struempell-Marie	G.C. Arthritis	Rheumatic Syndrome	Tubercular Arthritis
37%	28%	17%	11%	4.5%	2%	.5%

TYPE 1—83 Patients

N.S.T. Group 50% A.S.T. Group 50%

	Male	Female	Tissue Change	Active	Fever	Clinical Heart	Radio. Sclerosis	Sugar	Nephropathies	Tonsil	Teeth	Antra
N.S.T.	31%	69%	38%	50%	30%	12%	18%	2%	12%	38%	7%	5%
A.S.T.	20%	80%	59%	56%	50%	20%	11%	17%	22%	39%	12%	5%

Age: In N.S.T. group, highest percentage, 48 per cent under 30 years; 43 per cent 31-40 years.

In A.S.T. group, highest percentage, 44 per cent 31-40 years; 22 per cent under 30 years, and another 22 per cent from 41 to 50 years.

Duration: In N.S.T. group, highest percentage, 24 per cent with duration of months; 24 per cent with duration 6-10 years.

In A.S.T. group, highest percentage, 17 per cent with duration 2 years; 17 per cent with duration 6-10 years.

Comment: In type 1, the A.S.T. group show a definitely higher percentage showing tissue change, fever, clinical heart, sugar in urine, nephropathies and infected teeth. *Apparently the actual duration of the arthritis is of less importance than is the individual reaction to remission and relapse of the disease.*

TYPE 2—61 Patients

	N.S.T. Group 31%			A.S.T. Group 69%			
	Tissue Change	Active	Fever	Clinical Heart	X-Ray Sclerosis	Sugar Urine	Nephropathies
N.S.T.	11%	21%	11%	11%	80%	—	16%
A.S.T.	26%	33%	26%	45%	54%	12%	24%

	Elevated Blood Pressure	Tonsils	Teeth	Antra	Male	Female
N.S.T.	21%	84%	31%	5%	68%	32%
A.S.T.	29%	71%	7%	7%	48%	52%

Age: In the N.S.T. group, highest percentages, 42 per cent from 51-60; 21 per cent 41-50 years, and 21 per cent 61-70 years.

In the A.S.T. group, highest percentages, 45 per cent from 51-60 years; 27 per cent from 61-70.

Duration: In the N.S.T. group, highest percentages, 26 per cent with duration of months; 21 per cent with duration 5 years.

In the A.S.T. group, highest percentages, 21 per cent duration of months; 19 per cent duration 6-10 years.

Comment: In Type 2, the A.S.T. group again show a definitely higher percentage with tissue change, fever, clinical heart, sugar in urine and nephropathies; but the N.S.T. group show the higher percentage of infected teeth.

MIXED TYPE—37 Patients

	N.S.T. Group 30%			A.S.T. Group 70%			
	Tissue Change	Active	Fever	Clinical Heart	X-Ray Sclerosis	Sugar Urine	Nephropathies
N.S.T.	54%	18%	18%	45%	55%	—	27%
A.S.T.	35%	46%	54%	35%	55%	23%	31%

	Elevated Blood Pressure	Tonsils	Teeth	Antra	Male	Female
N.S.T.	27%	82%	—	18%	54%	46%
A.S.T.	15%	73%	19%	12%	35%	65%

Age: In the N.S.T. group, highest percentages, 64 per cent from 41-50 years; 36 per cent from 51-60.

In the A.S.T. group, highest percentages, 38 per cent from 51-60 years; 27 per cent from 41-50.

Duration: In the N.S.T. group, highest percentage, 45 per cent with duration of months.

In the A.S.T. group, highest percentage, 31 per cent with duration from 6-10 years.

Comment: In contrast to the findings in Types 1 and 2, the A.S.T. observations in the Mixed Type show a reversal of percentages. In the Mixed Type, the N.S.T. group show the higher percentage with tissue change, clinical heart and elevated blood pressure; while the A.S.T. group continue to show the greater number with activity, fever, sugar in the urine, nephropathies and infected teeth.

STRUEMPELL-MARIE TYPE—24 Patients

N.S.T. Group 58% A.S.T. Group 42%

	Male	Female	Tissue Change	Active	Fever	Clinical Heart	X-Ray Sclerosis	Sugar Urine	Nephropathies	Tonsil	Teeth	Antra
N.S.T.	86%	14%	50%	57%	28%	28%	8%	—	14%	50%	7%	—
A.S.T.	80%	20%	50%	70%	20%	10%	17%	20%	20%	20%	20%	—

Age: In the N.S.T. group, highest percentages, 57 per cent under 30 years; 36 per cent from 31-40.

In the A.S.T. group, highest percentages, 50 per cent under 30 years; 50 per cent from 31-40.

Duration: In the N.S.T. group, highest percentage, 57 per cent duration 6-10 years.

In the A.S.T. group, highest percentage, 30 per cent duration 6-10 years.

Comment: In this group, the A.S.T. show a higher percentage with active arthritis, sugar in urine, nephropathies and infected teeth; whereas the N.S.T. group show more with fever, clinical heart and definitely more with tonsils.

Gonorrheal Arthritis—11 patients—N.S.T. 45 per cent; A.S.T. 55 per cent.

Rheumatic Syndrome—5 patients—N.S.T.—60 per cent; A.S.T. 40 per cent.

Tubercular Arthritis—1 patient—N.S.T. 100 per cent.

TISSUE CHANGE

Ten patients, or 5 per cent of the 222 arthritics, were obese. Eighty-five, or 39 per cent, showed marked loss of weight. Of the latter group, 39 per cent were in the N.S.T. Group and 61 per cent in the A.S.T. Group.

WEIGHT-LOSS GROUP—85 Patients

	Type 1	Type 2	Mixed Type	Struempell-Marie	Gonorrheal Arthritis	Rheumatic Syndrome	Tubercular Arthritis
N.S.T.	45%	7%	18%	21%	3%	3%	3%
A.S.T.	46%	21%	18%	10%	4%	1%	—

	Active	Fever	Clinical Heart	X-Ray Sclerosis	Sugar Urine	Nephropathies
N.S.T.	55%	43%	12%	29%	—	21%
A.S.T.	60%	48%	35%	29%	15%	29%

	Elevated Blood Pressure	Tonsils	Teeth	Antra	Male	Female
N.S.T.	3%	36%	3%	3%	51%	49%
A.S.T.	10%	40%	18%	8%	46%	54%

Age: In the N.S.T. group, highest percentages, 39 per cent under 30 years; 33 per cent from 31-40.

In the A.S.T. group, highest percentages, 35 per cent from 31-40 years; 20 per cent under 30.

Duration: In the N.S.T. group, highest percentages,
24 per cent with duration 6-10 years;
18 per cent with duration 2 years.

In the A.S.T. group, highest percentages,
20 per cent with duration 2 years;
18 per cent with duration 6-10 years.

Comment: In this group of arthritics with marked weight loss approximately the same percentage of group N.S.T. and A.S.T. appear in the different types of arthritis, except in Type 2, which showed a much higher percentage of A.S.T. than N.S.T. Disregarding type of arthritis, the A.S.T. group with weight loss showed higher percentage of clinical heart, sugar in urine and infected teeth.

BLOOD PRESSURE

Thirty, or 14 per cent of the entire group, showed elevated blood pressure. Of this 30, 33 per cent were in the N.S.T. group; 67 per cent in the A.S.T. group.

Clinical Heart—60 patients—27% of entire group
N.S.T. Group 35% A.S.T. Group 65%

*X-Ray Sclerosis**—72 patients—37% of those on whom x-rays were taken
N.S.T. Group 44% A.S.T. Group 56%

*Arteriosclerosis comparisons have not been stressed in this paper, as a study of Arteriosclerosis in Arthritis is now in the process of preparation.

Sugar in Urine—23 patients—10% of entire group
N.S.T. Group 4% A.S.T. Group 96%

Nephropathies—44 patients—20% of entire group
N.S.T. Group 34% A.S.T. Group 66%

Active—100 patients—45% of entire group
N.S.T. Group 39% A.S.T. Group 61%

Fever—76 patients—34% of entire group
N.S.T. Group 31% A.S.T. Group 69%

Comment: In the patients showing clinical hearts, sugar in urine, nephropathies, activity of arthritis, fever and elevated blood pressure, a definitely higher percentage occurs in the A.S.T. group than in the N.S.T. group.

Age

Twenty-two per cent of the entire group were under 30; 27 per cent between 31 and 40; 19 per cent between 41 and 50; 21 per cent between 51 and 60; 9 per cent between 61 and 70; and 2 per cent over 70.

	Under 30 yr.	31-40 yr.	41-50 yr.	51-60 yr.	61-70 yr.	Over 70 yr.
N.S.T.	71%	43%	38%	28%	20%	40%
A.S.T.	29%	57%	62%	72%	80%	60%

Comment: The N.S.T. group showed the higher percentage only in the youngest group of patients, that is, in those under thirty years of age. The A.S.T. group showed the higher percentage in each of the succeeding decades.

Sex

	Males 99, or 45 per cent of entire group	Females 123, or 55 per cent
N.S.T.	51%	36%
A.S.T.	49%	64%

Focal Infection

	Tonsils (125—56%)	Teeth (29—13%)	Antra (14—6%)
N.S.T.	44%	38%	43%
A.S.T.	56%	62%	57%

Finally, as to the possible prognostic value of the sugar tolerance test: A comparison of the functional status, from an arthritic point of view, of those of the entire group followed three years or over, was made, and the following points brought out:

One hundred and forty-five patients were observed over this period. At the present time 31, or 21 per cent, are dead; 56, or 39 per cent, lead fairly normal lives with no joint handicap; 25, or 17 per cent, are ambulatory, showing moderate crippling; and 33, or 23 per cent, show marked joint crippling and disability. Dividing this group as they had reacted to the sugar tolerance test while in the hospital we have:

	No Joint Handicap	Ambulatory	Marked Crippling	Dead
N.S.T.	50%	50%	33%	56%
A.S.T.	50%	50%	67%	64%

Of those who went on to marked crippling, it is interesting to note that 13 of the 23 who had shown *abnormal* sugar tolerance had also shown marked loss of weight; while 3 of the 10 who had shown *normal* sugar tolerance had shown weight loss. Tonsils and teeth as foci of retained infection are present in higher percentages in the A.S.T. group than in the N.S.T. group. No other marked difference as regards disease complications were noted between the N.S.T. and A.S.T. groups. Therefore, it appears that a natural query is, if there is no distinct difference in complicating diseases as such in either the N.S.T. or A.S.T. group, may there be a greater dependence or relationship to the disturbances of the homeostatic systems and less to the disease entities and focal infections than heretofore has been proved? Another interesting observation is that if the dead and those showing marked crippling are combined, there are 64 out of the 145 who may be considered to belong to our "problematical" group of arthritics. Forty-three, or 68 per cent, of this last group had shown abnormal sugar tolerance, while 20, or 32 per cent, had shown a normal tolerance.

Serial Sugar Tolerance Tests

A small group of 33 patients were given sugar tolerance tests repeatedly over periods of from one to nine years. Twenty-two of this group showed tests which remained in the A.S.T. group constantly, irrespective of activity or inactivity of arthritis, or of any other systemic variation.

Of the twenty-two, fourteen had at no time positive tests for sugar in any of the routine urinalyses during the entire period they were observed. Five of the fourteen did show a slight trace of sugar in the urine during the sugar tolerance test, at the time when the blood showed a reading above 200. This point is of particular interest. Why in this group of arthritics does the threshold of leakage of sugar through the urine not appear until the blood sugar is 200 or over, when in the average person it is expected when the blood sugar reaches 180 mg.? In this group certainly a marked disturbance in the homeostatic control of the blood sugar is suggested. Eight patients out of the twenty-two who continued in the A.S.T. group over the years showed at various times questionable signs of sugar in routine urinalyses.

None of the eight, however, showed constantly sufficient sugar to classify them as diabetics. Eleven of the thirty-three in this group with serial tolerance tests showed abnormal curves in the first test but normal, or very slightly abnormal, curves in the last test. No outstanding observations were made in this group.

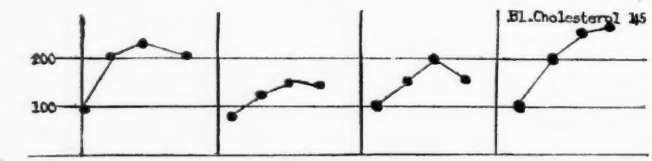
Comment: It is obvious from the above figures that an abnormal sugar tolerance individual does not become necessarily a normal one with cessation of activity of joints or with the subsidence of infection. Also an abnormal sugar tolerance over a period of years is not indicative of potential diabetes in this group.

A summary of the medical history of one of the cases so followed may be of interest. This individual was a woman first seen at the Robert Brigham Hospital at the age of 47, six years after the onset of her arthritis. Her family tissue inheritance was fair. Her married history was irrelevant as given except for great emotional strain over a period of years. The past history was essentially negative except for constipation of long standing. It took ten years from the onset of fleeting pains about the region of the joints for her arthritis to progress to marked deformity of the hands, flexion contraction of the knees with limited motion, and moderate involvement of the ankles. At the time of her admission to the hospital she had not walked for two years. On admission, the only positive physical findings were the presence of abnormal tonsils and limitation of joint motion with deformity. Radiograms showed destruction of cartilage, narrowing of joint spaces, bone absorption and thinning. This patient has been followed in and out of the hospital for ten years. During this time posterior capsuloplasty was performed on both knees with subsequent marked improvement in her functional activity. Her basal metabolism remained practically within normal limits; her vital capacity about half normal. At the present time she is able to lead a fairly normal life; walking without support, and with very little difficulty; doing her own housework. The last x-rays showed increased calcium deposition in the bones. During the entire period of observation there has been no demonstrable activity of the arthritis. This patient has carried her tonsils throughout this time and still has them at the age of 57 after 16 years' duration of her arthritis. There has been present at times localized infection of the tonsils, during one such attack her blood sedimentation rate was somewhat high, but at this time her white count was only 8,300. However, in spite of these tonsils the patient has not shown general systemic reaction to bacteria or infection. The striking point in this chart is that while the urine remained negative throughout the years, except for the small amount of sugar shown during the test with the high blood sugars, the sugar tolerance test remained abnormal, and the last test showed the lowest tolerance of any recorded.

To sum up the study of the arthritic. There is no question but that the general statement is basically correct that the arthritic is not merely a matter of joint pathology. There is, besides disturbance of the fluid content of the body, disturbance of the water and salt content

SUGAR TOLERANCE CHART—CASE NO. 333

TYPE 1 ARTHRITIS



Blood—	96-205-233-207	80-128-154-147	100-156-200-167	101-200-256-270
Urine—	No report	No report	No report	0-0-.30%-.33%
	1924 April	1925 January	1925 April	1933 August
Age	48 years	49 years	49 years	57 years
Duration	7 years	8 years	8 years	16 years
Weight	Normal—130 lbs.	No marked change	No marked change	No marked change
Activity	Inactive	Inactive	Inactive	Inactive
Blood Pressure	Low	Low	Low	Low
Clinical Heart	Negative	Negative	Negative	Negative
Urine	Negative	Negative	Negative	Negative
Foci Infection	Tonsils	Tonsils	Tonsils	Tonsils
Temperature	Normal	Normal	Normal	Normal
X-Ray Arteries	No sclerosis	No sclerosis	No sclerosis	No sclerosis

of the blood; there is disturbance of the normal homeostasis* of blood sugar, protein, fat, calcium and phosphorus. The maintenance of adequate oxygen supply to the tissues is altered. There is a question as to the degree of alteration of the homeostasis of blood neutrality. There is definite alteration of the PH content of tissue. There is definite alteration in the constancy of body temperature and basal metabolism. There is no question about the disturbance in the general functions of the sympathetic and parasympathetic nervous systems. There are definite changes in the general features of body stabilization.

During the past two years' work with the Robert Brigham Hospital Survey of Chronic Disease, this definite impression, though not a measurable fact, has been noted. In an arthritic in Type 1, or in the Mixed Type Groups, a history of marked loss of weight, activity of joints, febrile chart, easy reaction to infection, clinical heart change from normal, variations from normal in urinalyses, a history of emotional instability, or evidence of an unstable nervous system, plus an abnormally low sugar tolerance, are indications of a poor prognosis. The addition of poor inheritance and poor environment to the other poor prognostic signs may be the deciding factors tipping the scale downward for the individual to whom they apply. These may be the factors which produce his disastrous reaction to bacteria, toxin, or allergy, which the more fortunate arthritic overcomes, or handles with less eventual destruction of bone, joint and tissue. The disturbances of the various portions of the body functions are so widespread in their distribution and character in arthritis that a natural query is: May the etiology of arthritis lie in disturbed functions of the pancreas, liver or adrenals without a diabetic syndrome being present?

SUMMARY

1. A study of the sugar tolerance in 222 arthritics was made. Fifty-seven per cent of these patients showed an abnormal tolerance as contrasted to 43 per cent with normal tolerance. Careful analyses from the point of view of sex, age, duration of arthritis, activity of arthritis, fever, elevated blood pressure, tissue change, arteriosclerosis, demonstrable foci of infection, and heart and urinary changes showed no *marked* difference between the normal and abnormal sugar tolerance groups. Those with normal sugar curves and those with abnormal showed a certain amount of infection, activity, fever and systemic abnormalities. There was a slightly higher percentage of these changes in the abnormal group than in the normal.
2. Forty-eight patients were in the AGE GROUP under 30 years. Seventy-one per cent of these showed a normal sugar tolerance. In each succeeding age group, the majority fell in the abnormal sugar tolerance group.
3. Of the 85 patients with marked loss of weight, those with abnormal sugar tolerance showed a forty per cent higher incidence of activity of arthritis, of fever, and of abnormal hearts and urines than did those with normal sugar tolerance.
4. One hundred and forty-five of the 222 arthritics have been under observation for three or more years. A comparison of their functional status at the present time showed that those with no joint handicap and those with mild joint involvement are divided equally between the normal and abnormal sugar tolerance groups. It seems important to emphasize that the combined groups, comprised of those who have died or who have progressed to more extensive joint involvement and marked crippling, showed 68 per cent who had had abnormal sugar tolerance tests as contrasted with 32 per cent with normal tests. Therefore, it appears that a low sugar tolerance is of prognostic value in a patient with Type 1 or Mixed Type arthritis, if such a patient gives a history of marked weight loss and shows abnormal heart function and urinary changes, and we believe such prognosis to be poor.
5. Serial sugar tolerance tests covering one to nine years on a group of 33 patients showed that even a markedly low sugar tolerance does not per se indicate a future diabetic. Instances are given of sugar tolerances remaining definitely low over a period of nine years without the appearance of the slightest signs or symptoms of diabetes. These serial tests have also shown that in an arthritic the low sugar tolerances do not always return to normal at the time of cessation of activity of the arthritis, or in the absence of demonstrable infection.
6. Because of the multiple involvement of the body defenses in the "problematical group" of arthritics, the query arises as to whether the etiology of the disease may not lie in change in function of the pancreas, liver, adrenals, or pituitary affecting the autonomic control of the body.

*Cannon, Walter B.: "The Wisdom of the Body." Page 24: "The coördinated physiological processes which maintain most of the steady states in the organism are so complex and so peculiar to living beings—involving, as they may, the brain and nerves, the heart, lungs, kidneys and spleen, all working coöperatively—that I have suggested a special designation for these states, *Homeostasis*. . . . It means a condition—a condition which may vary, but which is relatively constant."

MEDICAL PROGRESS

CARDIOVASCULAR REVIEW FOR 1932

BY PAUL D. WHITE, M.D.*

(Continued from last week)

Bacterial endocarditis: *Acute.*—Dickar has reported 2 cases of acute bacterial endocarditis caused by the *Bacterium acidilactici.*

Lord has studied 23 cases of pneumococcus endocarditis autopsied at the Massachusetts General Hospital. In 8 of these 23 cases, there was chronic as well as acute endocarditis. The left side of the heart alone was involved in 18 cases, the right alone in 3, and both sides in 2. The mitral valve was affected in 13 instances, the aortic in 12, and both in 5. The tricuspid valve was involved in 5, including one with involvement of the pulmonary valve also. Lord has known of no definitely established case of recovery. Fourteen of 337 cases of lobar pneumonia autopsied between 1898 and 1929 showed acute endocarditis (4.15 per cent).

Phipps has reported an analysis of 44 cases of acute bacterial endocarditis autopsied at the Boston City Hospital from 1914 to 1929, exclusive of pneumococcus endocarditis. Thirty-two were males and 12 were females. The commonest age was forty-five to fifty years. In about 60 per cent of the cases there was evidence of old pathology in the endocardium, particularly arteriosclerosis. The organism most commonly responsible was the *Streptococcus hemolyticus*, in 21 of the 44 cases. Second was the *Staphylococcus aureus* in 11 cases, third the *B. coli communis* in 5 cases, while infrequently were found the gonococcus, 2 cases, meningococcus, 2 cases, staphylococcus albus, 1 case, *Streptococcus hemolyticus* plus *Staphylococcus aureus*, 1 case, and *B. coli communis* plus *Staphylococcus aureus*, 1 case. The duration of life ranged from three days to three weeks.

Dickar, L.: Acute bacterial endocarditis due to bacterium acidi-lactici. Arch. Int. Med. 49:788, 1932.

Lord, F. T.: Pneumococcus endocarditis. New Eng. J. Med. 207:767, 1932.

Phipps, C.: Acute bacterial endocarditis. New Eng. J. Med. 207:768, 1932.

Subacute.—Morrison has analyzed a large series of cases of subacute bacterial endocarditis seen in six Boston hospitals in the four years, 1928 to 1931 inclusive. There was a total of 177 such cases, while during the same period of time in the same hospitals there were 772 cases of rheumatic fever; the ratio was about 3 to 10, except at the Children's Hospital where the ratio was only 4 to 100. The *Streptococcus viridans* was responsible for the great majority of cases (113 out of 135 with positive blood cultures). Out of 203 cases 134 were rheumatic in background, 11 were congenital, 3 luetic, and 1 arteriosclerotic. The mitral valve alone was involved in 25 cases, the aortic alone in 18, both valves in 38, and other valves in 8.

Morrison, H.: Subacute streptococcus endocarditis. New Eng. J. Med. 207:770, 1932.

Cardiovascular syphilis.—Stadler has written a small book on syphilis of the heart and blood vessels, giving in an interesting way an analysis of statistics concerning cardiovascular syphilis found in the literature, and concluding with chapters on prognosis and treatment. He believes that in spite of occasional or even frequent disappointments, fairly vigorous antiluetic therapy should be carried out in most cases beginning with bismuth (in preference to mercury) and going on to arsenic, with iodide given while these courses are going on. He advises two full courses the first year, and after that one course a year. Early intensive arsenic treatment of primary syphilis has not seemed to decrease the later incidence of aortitis; some statistics suggest that it causes an increase.

Moore, Danglade and Reisinger have studied syphilitic aortitis uncomplicated by aortic regurgitation or aneurysm and found in the twenty-year period from 1910 to 1930 at the Johns Hopkins Hospital 105 patients who showed this condition at postmortem examination. The clinical diagnosis was correctly made in only 4 of these 105 cases; in 13 more something was suspected to be wrong with the aorta. An additional 35 of the 105 cases might have been correctly diagnosed if careful attention had been paid to all symptoms and signs. Thirty-four of the series died with hearts and aortas thought clinically to be normal. Hypertension was an infrequent accompaniment of syphilitic aortitis. The Wassermann reaction was positive in 75 per cent of the cases. Seven suggestive symptoms and signs are (1) roentgen-ray evidence of aortic dilatation, (2) increased retrosternal dulness, (3) dyspnea on exertion, (4) tympanitic aortic second sound, (5) progressive heart failure, (6) substernal pain, and (7) paroxysmal dyspnea.

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Moore, Danglade and Reisinger have also published an important paper on the subject of the treatment of cardiovascular syphilis, based on studies of 53 patients with aortic aneurysm and 112 with aortic regurgitation. "Not one of 117 patients with early syphilis who received three or more courses of arsphenamine, and treatment with mercury during periods between the courses, presented any evidence of cardiovascular involvement during the period of observation, while 24 of 285 patients who had received less than this amount of treatment were observed to acquire syphilitic aortitis, aneurysm, or aortic regurgitation. Adequate treatment for early syphilis almost certainly protects the majority of patients so treated against subsequent cardiovascular syphilis. Of 6,420 patients from our own clinic with various forms of late syphilis, 10 per cent had cardiovascular syphilis (clinically). Of the total number 2.7 per cent had aortic regurgitation and 1.2 per cent aortic aneurysm. . . . Cardiovascular syphilis is twice as common in males as in females and about twice as common in the Negro as in white patients. . . . The majority of cases of aneurysm and aortic regurgitation occur in the fifth decade of life. . . . The average interval of time between infection and the development of cardiovascular symptoms is about twenty years. . . . The actual incidence of complicating neurosyphilis was about 35 per cent (concealed in half). . . . Eighty-nine per cent of our patients had never received any treatment for syphilis before the development of cardiovascular syphilis. Of the remainder not one had received adequate treatment for early syphilis." The authors describe their method of cautiously using mercury, bismuth, the iodides, neoarsphenamine, and bismarsen in small doses over periods of years as follows: Begin with intramuscular injection of insoluble bismuth salt, 0.1 gram every four or five days for a few weeks, then give 0.2 gram once a week until a total interval of ten to twelve weeks on bismuth therapy has elapsed; give simultaneously with the bismuth potassium iodide or sodium iodide by mouth, 1.3 grams three times daily, increasing to 4 grams three times daily. At the end of this period of ten to twelve weeks, change to neoarsphenamine or bismarsen (bismuth arsphenamine sulphonate) 0.05 to 0.1 gram once a week, increasing to maximum dose of 0.3 gram (rarely 0.45 to 0.6 gram) for ten to twelve weeks, the bismarsen to be given to the less favorable cases; after the bismarsen or neoarsphenamine course, return at once without pause to the insoluble bismuth and iodide therapy; alternate these two courses over a minimum time interval of two years; then institute long periods of rest with an occasional course of bismuth followed by one of bismarsen or neoarsphenamine, averaging once a year for the duration of life.

The authors have obtained the following results by this therapy: Fifteen cases of well-treated aortic aneurysm gave 40 per cent mortality, with average duration of life of seventy-five months, as compared with a mortality of 90 per cent in the same interval of time and a duration of life of only nineteen months for 22 cases of aortic aneurysm receiving little or no treatment. Twenty-five cases of well-treated luetic aortic regurgitation gave a mortality of 16 per cent and duration of life of seventy-one months, as compared with 91 per cent mortality and three months' duration of life for 57 cases receiving little or no treatment. Out of 56 surviving cases of this treated series, 21 were symptom-free and able to work, 26 had persistent symptoms but could carry on light work, and 9 were incapacitated. Fifty-seven of the whole group of 165 cases died of progressive cardiac failure, 28 (10 cases of aneurysm and 18 of aortic regurgitation) died suddenly, 11 died of unknown cause, and 13 died of other than cardiovascular causes. A fixed positive Wassermann reaction is the rule in cardiovascular syphilis and so may be completely disregarded.

The authors conclude from the analysis of their material "that properly supervised antisyphilitic treatment, with adequate general medical care, given to patients with aneurysm or aortic regurgitation, prolongs life, alleviates symptoms, maintains the ability to pursue a gainful occupation, and reduces the period of incapacity."

Blackford and Boland have reported their experience in the treatment of 100 cases of aortic syphilis. Twenty-five out of 28 carefully studied cases had pain and this pain was relieved in all but one case by the intramuscular injection of sodium bismuth tartrate (routinely 2.0 cc. of a 1.5 per cent solution twice a week in courses of ten injections). "This drug can be administered over long periods with good results. Fifty patients with heart disease associated with syphilis were also treated with bismuth. Marked clinical improvement was usually noted. Congestive heart failure in cardiovascular syphilis urgently demands the immediate administration of bismuth; general measures for the treatment of the failing heart are not enough." Salyrgan as a diuretic may help a good deal. Potassium iodide is to be given orally also.

Blackford, L. M., and Boland, J. H.: Bismuth in treatment of cardiovascular syphilis. J. A. M. A. 99:1902, 1932.

Moore, J. E.; Danglade, J. H., and Reisinger, J. C.: Diagnosis of syphilitic aortitis uncomplicated by aortic regurgitation or aneurysm. Comparison of clinical and necropsy observations in one hundred and five patients. Arch. Int. Med. 49:753, 1932.

Moore, J. E.; Danglade, J. H., and Reisinger, J. C.: Treatment of cardiovascular syphilis: results obtained in fifty-three patients with aortic aneurysm and in one hundred and twelve with aortic regurgitation. Arch. Int. Med. 49:879, 1932.

Stadler, E.: Syphilis des Herzens und der Gefässe. Verlag von Theodor Steinkopff, 1932, 82 pages.

Other infections.— Place has written of his experiences with heart disease resulting from

diphtheria and scarlet fever, common and serious in the former, and rare and slight in the latter. He writes that in diphtheria, "prevention of the heart injury is entirely feasible. In over 700 cases of diphtheria occurring under observation in the hospital among the employees, no fatality has occurred in the last thirty-five years. No case of cardiac damage has appeared in patients receiving antitoxin treatment during the first twenty-four hours and only rarely when antitoxin was used during the second twenty-four hours."

Jones and Newsom have shown that the hearts of dogs, in which dental abscesses were produced experimentally, constantly suffered small vegetative or verrucose lesions on mitral or aortic valves or on both, as well as parenchymatous changes in the myocardium.

Jones, N. W., and Newsom, S. J.: Experimentally produced focal (dental) infection in relation to cardiac structure. Arch. Path. 13:392, 1932.
Place, E. H.: Heart in diphtheria and scarlet fever. New Eng. J. Med. 107:864, 1932.

The Heart in Thyroid Disease: *Thyrotoxicosis.*—Anderson has reported a study of 426 patients with auricular fibrillation in thyrotoxicosis, an incidence of 6 to 9 per cent. Quinidine was successful in restoring normal rhythm following thyroidectomy in 90 to 96 per cent of the cases.

Barker, Bohning and Wilson have published a study of 108 cases of auricular fibrillation in Graves' disease, an incidence of approximately 15 per cent. The average age of the patients studied was 51.5 years and the average basal metabolic rate before treatment plus 48 per cent. The arrhythmia is likely to be transient. Digitalis is less effective than with other etiological factors behind the auricular fibrillation and the tolerance to the drug is greater (134 per cent of the theoretical normal). Normal rhythm is restored by operation, with or without quinidine therapy afterwards in 90 per cent of the cases. The average weight of the hearts of 13 patients coming to autopsy was 438 grams (the two heaviest hearts weighed 530 grams each).

Kepler and Barnes have found enlargement of the heart (increased weight) in 49 per cent of 89 fatal cases of hyperthyroidism. Severe congestive failure occurred in 27 out of 178 fatal cases of hyperthyroidism, to be attributed to that factor alone in 9.

Lev and Hamburger have reported 3 new cases of angina pectoris and hyperthyroidism relieved of their heart pain following the administration of iodine and thyroidectomy.

Markowitz and Yater have found that fragments of heart muscle removed from two-day chick embryos, before the appearance of nerve elements, react vigorously to thyroxin with progressively greater increase in rate of pulsation, leading sometimes to fibrillation and paralysis.

Weller and his associates have made a morphological study of the hearts of 35 patients with exophthalmic goiter and found, with few exceptions, "no gross or microscopical changes not equally represented in a carefully matched control series. The exceptions found were (1) a relatively higher incidence of myocardial fibrosis (80 per cent compared to 51.5 per cent in the control series), endocardial sclerosis, and cellular infiltrations in the series with exophthalmic goiter, and (2) one case in the series of 35 which showed an active focal myocarditis for which no etiological factor could be ascertained other than the hyperthyroid state."

Anderson, J. P.: The incidence of auricular fibrillation (in thyrotoxicosis) and results of quinidine therapy. Am. Heart J. 8:128, 1932.
Barker, P. S.; Bohning, A. L., and Wilson, F. N:. Auricular fibrillation in Graves' disease. Am. Heart J. 8:121, 1932.
Kepler, E. J., and Barnes, A. R.: Congestive heart failure and hypertrophy in hyperthyroidism: A clinical and pathological study of 178 fatal cases. Am. Heart J. 8:102, 1932.
Lev, M. W., and Hamburger, W. W.: Studies in thyroid heart disease. Angina pectoris and hyperthyroidism. Am. Heart J. 8:109, 1932.
Markowitz, C., and Yater, W. M.: Response of explanted cardiac muscle to thyroxine. Am. J. Physiol. 100:162, 1932.
Weller, C. V.; Wanstrom, R. C.; Gordon, H., and Bugher, J. C.: Cardiac histopathology in thyroid disease. Preliminary report. Am. Heart J. 8:8, 1932.

Myxedema.— Fahr has reported a study of 17 cases of myxedema. Seventy-five per cent showed signs and symptoms of heart failure in varying degree, 30 per cent severely. The volume of the heart in the severe cases was increased approximately 100 per cent. "The age of onset of myxedema heart varied between nineteen and seventy years of age. The average age was forty-five at onset."

Fahr, G.: Myxedema heart. A report based upon a study of 17 cases of myxedema. Am. Heart J. 8:91, 1932.

The Heart in Hypertension.—Blackford and Wilkinson have concluded from a ten-year study of 202 cases of hypertension, that "hypertension is twice as frequently found in women as in men," that "the mortality rate after ten years is twice greater in men," that "hypertension results from a constitutional hypertonicity of the autonomic neurovascular control in the large majority of instances and is a compensatory angiospasm in the others," that "treatment of uncomplicated hypertension is a matter of mental and physical hygiene rather than of drugs," that "treatment of late results of hypertension requires skillful use of medical and physical measures, added to psychotherapeutic measures," and that "the physician who is a good and cheerful psychologist will be the most successful in relieving the symptoms of hypertensive cardiovascular disease."

Murphy, Grill, Pessin, and Moxon have reported the study clinically and morphologically of 375 cases of essential (primary) hypertension which came to postmortem examination. They conclude that "unifying features of all stages are hypertension, cardiac hypertrophy,

and arteriosclerosis." The chief complications were heart failure (cause of 50 per cent of the deaths), infections (14.2 per cent of the deaths), apoplexy (13.4 per cent of the deaths), and renal failure (10.4 per cent of the deaths). "Arteriosclerosis of the renal vessels is a constant and characteristic feature of essential hypertension at autopsy." "Histologically the principal lesion is an arteriosclerosis. The wide variety of symptoms and signs appears to depend upon the extensiveness, the rate of progress, the severity, and the location of the arteriosclerosis. The clinical and histological observations on Group 3 (so-called malignant hypertension) differ from those in Group 2 (benign) only in degree. All of the symptoms and histological lesions observed, we believe, may be produced by arteriosclerosis alone." The heart weights were below 400 grams in only 18.13 per cent of all the cases.

Wiggers has written that "careful evaluation of the experimental and clinical evidence leads to the conclusion that the circulatory changes in clinical hypertension are usually produced by the combined effects of increased peripheral resistance and decreased elasticity of the aorta." He adds further, "The question may now be raised whether it is advisable to lower blood pressures in hypertension unless real dangers appear to be impending. . . . I have repeatedly demonstrated experimentally, for example, that amyl nitrite, contrary to general belief, reduces blood flow through the coronary and cerebral vessels. Obviously such lowering of blood pressure defeats the purpose of natural compensatory mechanisms, and at times may actually be dangerous."

Stieglitz considers that the degree to which the diastolic blood pressure approaches normal on the administration of nitrites may be taken as an index of the ability of the arteries to relax in cases of essential hypertension. Thus, he considers the test a valuable one for prognosis.

Elberg and Skulskiy have studied the effect of calcium thiocyanate therapy in 63 patients with hypertension. The drug was given in doses of 0.05 gram (1 grain) three times daily after meals and continued for periods of eight weeks. Favorable results occurred and lasted sometimes for ten months; success was noted in 90 per cent of the cases.

Goldring and Chasis have reported 74 trials of thiocyanate therapy in 50 patients, 46 with essential hypertension and 4 with chronic diffuse glomerulonephritis. The drug was 31 per cent effective in lowering blood pressure in the whole series, including success in 3 of the 4 patients with nephritis. Toxic manifestations occurred in 17 per cent of the total group, but in none of the 4 nephritic cases. Toxic symptoms consisted of psychosis in 6 cases, nausea in 3, fatigue without nausea in 1, dermatitis in 1, and collapse resulting in death in 2. The dosage found to be most effective in lowering blood pressure and least often attended by toxic manifestations was 0.326 gram (5 grains) given daily over a period of from fourteen to seventy-eight days. The authors conclude that the "uncertainty regarding proper dosage, coupled with the fact that frequently there is slight if any difference between the amounts that produce therapeutic and toxic effects, lead us to believe that thiocyanate will have a much restricted usefulness in clinical medicine."

Palmer has reported treatment by potassium sulphocyanate of 35 well controlled patients, most of them showing the effects of continued arterial hypertension. "This drug when used in sufficient dosage caused a definite and marked lowering of the arterial blood pressure in 31 per cent of the patients." Sometimes toxic effects occurred, such as skin rashes, gastro-intestinal symptoms, and nervous excitement. Palmer states, however, that "toxic effects are reduced to a minimum by carefully controlled dosage." He believes that the drug may be of value in conjunction with a general régime of rest and diet.

Stieglitz believes that the long-continued administration of bismuth subnitrate has a favorable effect in spastic hypertonia and in angina pectoris. The dosage used is 30 grains a day or less.

Blackford, J. M., and Wilkinson, J. N.: Hypertension; study of two hundred two cases followed for an average period of ten years; with remarks on causes and treatment. Ann. Int. Med. 1:54, 1932.
Elberg, V. A., and Skulskiy, N. A.: Clinical aspect of thiocyanate therapy in the treatment of hypertension. Jurnal Medich. Tsikly, Kiev. 2:213, 1932.
Goldring, W., and Chasis, H.: Thiocyanate therapy in hypertension: II. Its effect on blood pressure. Arch. Int. Med. 49:934, 1932.
Murphy, F. D.; Grill, J.; Pessin, B., and Moxon, G.: Essential (primary) hypertension: A clinical and morphological study of 375 cases. Ann. Int. Med. 6:31, 1932.
Palmer, R. S.: The hypotensive action of potassium sulphocyanate in hypertension. Am. J. Med. Sc. 184:473, 1932.
Stieglitz, E. J.: Prognostication in hypertensive arterial disease. Illinois M. J. 62:414, 1932.
Stieglitz, E. J.: Therapeutic results with bismuth subnitrate in hypertensive arterial disease. J. Pharmacol. & Exper. Therap. 46:343, 1932.
Wiggers, C. J.: Physical and physiological aspects of arteriosclerosis and hypertension. Ann. Int. Med. 6:12, 1932.

The Cor Pulmonale.—Achard has reported 3 cases of so-called Ayerza's disease—the "black cardiacs", in which the pulmonary lesions resulting in the extreme cyanosis were the effect of poison gas inhalation during the World War. Not one of these cases was syphilitic. Achard also points out the fact that this condition was known many years before Ayerza appended the new designation, "*cardiacos negros.*"

Achard, C.: La Maladie des Cardiaques Noirs et ses Rapports avec l'Intoxication par les Gaz. Libman Anniversary Volumes. P. 39, 1932.

Coronary Disease.—Condorelli has published a small volume reviewing the anatomy, physiology, experimental pathology, and clinical as-

pects of the coronary circulation. This is a useful summary of data collected by many workers who are listed in a fairly complete bibliography.

Hochrein has written a comprehensive and valuable survey of the physiology and diseases of the coronary circulation in the form of a monograph of 227 pages. The anatomy and results of physiological and pharmacological experiments make up the first half of the book, while the second half takes up the pathological physiology, pathological anatomy, symptoms, signs, and treatment of coronary disease with especial reference to angina pectoris and coronary thrombosis. An extensive bibliography is appended.

Boas and Donner have made an analysis "of 615 males and females of the industrial class in New York City who were referred with the presumptive diagnosis of heart disease. (The patients were all Jews.) Eighty-eight per cent of the population from which the patients were drawn were under the age of fifty. Almost one-third of the 615 referred patients had disease of the coronary arteries. Of those with disease of the coronary artery, 71 per cent were under the age of 51. This represents an exceptionally high frequency of coronary artery disease, which is all the more remarkable in view of the preponderance of individuals under the age of fifty." "Our data," the authors say, "demonstrate that coronary artery disease, contrary to general belief, is more common, and occurs earlier in life, at least in certain groups of industrial workers" (of the Jewish race, should be added). They suggest the desirability of further studies along similar lines with different racial and occupational groups.

Condorelli, L.: Die Ernährung des Herzens und die Folgen ihrer Störung. Theodor Steinkopff, Dresden and Leipzig, 1932, 230 pages.
Boas, E. P., and Donner, S.: Coronary artery disease in the working classes. J. A. M. A. 98:2186, 1932.
Hochrein, M.: Der Coronarkreislauf. Physiologie. Pathologie. Therapie. 227 pages. 54 illustrations. Julius Springer, Berlin, 1932.

I. *Coronary occlusion.*—Barnes and Ball have reported their finding of gross myocardial infarction in 49 out of 1,000 unselected consecutive postmortem examinations. In 25 cases, the infarct was located in the anterior and apical portion of the left ventricle (supplied by the anterior descending branch of the left coronary artery), in 21 cases the infarct involved the posterior basal portion of the left ventricle (supplied generally by the right coronary artery), and in 3 cases both areas were affected.

Bartels and Smith have made a study "of the weights of the heart in 42 cases of cardiac infarction, in which all other known or supposed causes of cardiac hypertrophy have been excluded. Of the 42 cases, in 37 (88 per cent) there was definite gross cardiac hypertrophy. In 5 cases, the weights of the hearts were not above normal. The minimal increase in weight was 18 grams (9 per cent); the maximal increase, 341 grams (108 per cent), and the average increase, 132 grams (44 per cent). From the data given it would appear that cardiac infarction is a definite cause of cardiac hypertrophy."

Cataldi has reported the case of a man thirty-nine years old who died of heart failure and showed at postmortem examination intense luetic aortitis with complete occlusion of the right coronary artery at its mouth, aortic valve regurgitation, enormous hypertrophy of the left ventricle, and ventricular myocardial lesions, especially on the left side. There were macroscopic anastomoses between the left and right coronary arteries.

Crawford and his associates have studied electrocardiographic changes "in relation to the site of damage in thirty-four cats in which localized ventricular myocardial lesions had been produced by the electric cautery. Monophasic type curves were obtained which were classified as of the T_1 and T_3 types of Parkinson and Bedford. With almost complete consistency, lesions in similar sites produced the same type of curve. Lesions on the anterior surface of the left ventricle produced curves of the T_1 type, while those on the posterior surface of the left ventricle, including the apex, yielded the T_3 type. All right ventricular sites, except the base anterior in which only a slight change was induced, gave curves of the T_3 type. . . . In a few instances an R-T elevation was present in all three leads but as a rule to a greater extent in one lead. In three experiments, in each of which the lesion was located at the base anterior, depression rather than elevation of the R-T segment occurred."

Davis has reported that in a series of 76 cases of coronary thrombosis proved at postmortem examination in Chicago, only 36 (47 per cent) gave a history of pain, 29 (38 per cent) gave no history of pain, and 11 (15 per cent) were without history (sudden deaths).

Holland and Levine have reported that "in a study of 328 cases of coronary occlusion in which electrocardiograms were taken, there were 35 that showed essentially normal tracings at one time or another following the onset of attack.

"In 6 cases, so-called normal electrocardiograms were obtained from a few hours to seven days after the onset of attack. In all these, subsequent tracings showed characteristic alterations in the ventricular complexes. In 20 cases typical changes in the tracings taken soon after the onset of the attack were replaced by 'normal' curves at some subsequent time. . . . In 9 cases the only electrocardiograms obtained were normal. In only 1 of these 9 cases were curves obtained within the first ten days. Of the entire series of 328 cases there were only 8 in which 'normal' electrocardiograms were

obtained at any time during the first ten days."

Lisa and Ring have reported finding 100 cases of myocardial infarction or gross fibrosis among 942 necropsies performed at the City Hospital in New York from Jan. 3, 1928 to May 1, 1931. Thirty-two of the 100 cases showed definite recent scarring. Eighty-three were males; 17 were females. The average age was sixty-one years, the youngest twenty-eight, the oldest eighty-three. The average heart weight was 519 grams; the smallest heart of 200 grams was atrophied; the largest weighed 925 grams. The left ventricle and interventricular septum were affected in the great majority of cases. Of 90 of the cases, 75 (83 per cent) showed moderate to marked sclerosis, 8 slight sclerosis, and 7 no sclerosis of the coronary vessels. Coronary thrombosis was noted in 24 cases. Hypertension occurred in 60 per cent of the cases. Eleven died of angina pectoris and 12 of acute dyspnea.

Parkinson has published an excellent paper on coronary pain with a discussion of coronary thrombosis and of angina pectoris. He emphasizes the great variability of the seriousness of both conditions and refers to the overstressing of their dangers in the past.

Wolferth and Wood have reported 2 cases "to illustrate the use of Lead IV (an anteroposterior chest lead) in the electrocardiographic diagnosis of coronary occlusion. In Case 1, the three conventional electrocardiographic leads yielded little or no diagnostic information except on the fourth day. Nevertheless, for 8 days after the original infarction Lead IV showed striking and unmistakable deviation of the S-T interval from the isoelectric line. In Case 2, both Leads I and IV showed evidence of coronary occlusion. Lead IV showed a much more striking S-T interval deviation than was seen in Lead I.

"In a series of 33 controls deviation of the S-T interval from the isoelectric line was not seen."

Thus it is seen "that certain of the areas of the myocardium which are 'silent' in the three conventional leads have a definite effect on Lead IV."

Barnes, A. R., and Ball, R. G.: Incidence and situation of myocardial infarction in one thousand consecutive postmortem examinations. Am. J. M. Sc. 183:215, 1932.

Bartels, E. C., and Smith, H. L.: Gross cardiac hypertrophy in myocardial infarction. Am. J. M. Sc. 184:452, 1932.

Cataldi, G.: Sull'occlusione lenta delle arterie coronarie. (Contributo clinico ed anatomo-patologico.) Policlinico, 1932. (Sez. Med.) 39:152, 1932.

Crawford, J. H.; Roberts, G. H.; Abramson, D. I., and Cardwell, J. C.: Localization of experimental ventricular myocardial lesions by the electrocardiogram. Am. Heart J. 7:627, 1932.

Davis, N. S., III.: Coronary thrombosis without pain: its incidence and pathology. J. A. M. A. 98:1806, 1932.

Holland, C. W., and Levine, S. A.: Limitations of the electrocardiogram as an aid in the diagnosis of coronary occlusion. New Eng. J. Med. 207:545, 1932.

Lisa, J. R., and Ring, A.: Myocardial infarction or gross fibrosis: analysis of one hundred necropsies. Arch. Int. Med. 50:131, 1932.

Parkinson, J.: Coronary thrombosis. Brit. Med. J. 2:549, 1932.

Wolferth, C. C., and Wood, F. C.: The electrocardiographic diagnosis of coronary occlusion by the use of chest leads. Am. J. M. Sc. 183:30, 1932.

Miscellaneous Etiological Factors and Relationships: *Trauma* — Two cases of nonfatal stab wounds of the ventricles of the heart (Negroes) with clinical and electrocardiographic studies have been reported by Porter and Bigger who found coronary T waves in the electrocardiograms of both cases but no coronary pain. They believe that the actual severance of the muscle fibers which prevented normal contraction of this area of the myocardium accounts for the absence of pain which they assume to be due ordinarily to metabolites accumulating in muscle actively contracting but insufficiently supplied with blood.

Porter, W. B., and Bigger, I. A.: Nonfatal stab wounds of the ventricles, with electrocardiographic signs of coronary thrombosis and absence of angina pain. Am. J. M. Sc. 184:799, 1932.

Pregnancy — Carr and Palmer have found "that there is a definite progressive tendency toward left axis deviation in the electrocardiogram from the second to the sixth month (of pregnancy), while the axis remains about the same in the seventh and eighth months, definitely shifting back to the right in the ninth month." They further found that total inversion of Lead III in pregnancy was of no significance (it occurred in 29 of 264 instances).

Stander and Cadden have found that the cardiac output in normal pregnancy begins to rise above the normal level at the start of the fourth month and increases steadily until it is 50 per cent of the normal nonpregnant level at full term returning to its original level by the end of the third week of the puerperium.

Carr, F. B., and Palmer, R. S.: Observations on electrocardiography in heart disease associated with pregnancy, with especial reference to axis deviation. Am. Heart J. 8:238, 1932.

Stander, H. J., and Cadden, J. F.: Cardiac output in pregnant women. Am. J. Obst. and Gynec. 24:13, 1932.

The Heart in Obesity—Prodger and Dennig, after comparing various aspects of the circulation in 3 obese but otherwise healthy individuals (2 men and 1 woman) with the same aspects in 4 normal controls, have concluded that "the symptoms which are so commonly associated with cardiac insufficiency and which are frequently observed in cases of simple obesity are not due to an inefficient circulatory response, but rather to mechanical and chemical disturbances associated with obesity."

Prodger, S. H., and Dennig, H.: A study of the circulation in obesity. J. Clin. Investigation. 11:789, 1932.

Athletics and the Heart—Brooks has published a review of the so-called "athlete's heart." He finds that the cases formerly classed as the athlete's heart represent primary disease with subsequent relative overstress. Hypertrophy of the heart, such as may occur within physiological limits is so slight as to be undetectable by

our present methods of clinical study. Prevention of cardiac hypertrophy of pathological degree relies on the early recognition of cardiac disease and on the subsequent individual limitation of physical stress."

Steinhaus and his associates have measured biweekly roentgenographic records of the cardiac silhouette of 12 male dogs from 4 litters for area, greatest length, breadth, and transverse dimension. They have concluded from such studies that strenuous exercise (running and swimming) leads to an enlargement of the heart of growing dogs; this enlargement appears within three to five weeks from the beginning of exercise. "There are indications pointing to regression of the heart size when exercise is discontinued." There was little change of importance in the electrocardiograms of these exercised dogs. The trained dog tended to have a slower pulse and more respiratory arrhythmia than did the untrained dog.

Brooks, H.: The Heart of the Athlete. Libman Anniversary Volumes. P. 227, 1932.
Steinhaus, A. H., and associates. Studies in physiology of exercise: VIII. Chronic effects of running and swimming on hearts of growing dogs as revealed by roentgenography. IX. As determined electrocardiographically. Am. J. Physiol. 99:503, 1932.

The Heart in Old Age—Willius and Smith have found that only 48, or 12.6 per cent, of 381 persons who lived to be seventy years old or more died of heart disease.

Willius, F. A., and Smith, H. L.: Further observations on the heart in old age. A postmortem study of 381 patients, aged seventy years or more. Am. Heart J. 8:170, 1932.

Neuroses and Neurocirculatory Asthenia—Crile has reported relief of symptoms of neurocirculatory asthenia in most of the cases with this condition in which he has denervated the adrenal glands. Of 21 patients operated on with bilateral denervation in the past eighteen months, 18 have been much improved, another case has been well for four and one-half years after unilateral denervation and one other patient has been relieved of symptoms for fourteen years since a unilateral suprarenalectomy.

Crile, G. W.: Denervation of adrenal glands for neurocirculatory asthenia; technic and clinical results. Surg. Gynec. & Obst. 54:294, 1932.

PATHOLOGY

Myocardial disease.—Freeman and Griffin have reviewed the cases on record of spontaneous rupture of the heart and have found rupture of the right auricle in 35 cases, of the left auricle in 12 cases, of the right ventricle in 63 cases, of the left ventricle in 493 cases, and miscellaneous in 15 cases (mostly papillary muscles). They have reported 2 new cases, one a male aged sixty years, and the other a female aged seventy.

Ham has shown that "enormous single doses of irradiated ergosterol will produce massive calcification in the aorta, coronary vessels and cardiac musculature of the rat as soon as forty-eight hours after administration. Sections from the tissues twenty-four hours after administration show nothing that would presage such an imminent catastrophe so that the calcifications do not appear to depend on degenerative changes in the recipient tissues. On the other hand, the rapidity of formation, together with the massiveness of the calcifications, suggests very strongly that the prime factor in their causation is the inability of the serum to retain all its calcium in solution. It is suggested that precipitation depends on saturation of the serum with diffusible calcium plus other factors"—such as change in carbon dioxide tension and addition of other ions which would force a precipitation of calcium salts. "A marked inflammatory cell infiltration developed about the affected coronary vessels and about the calcified areas of cardiac muscle."

Freeman, W., and Griffin, E. D.: Cardiac rupture with perforation of interventricular septum. Report of two cases. Am. Heart. J. 7:732, 1932.
Ham, A. W.: Mechanism of calcification in the heart and aorta in hypervitaminosis D. Arch. Path. 14:613, 1932.

Endocardial Disease.—Anitschkow has published an important paper on studies he has made of atherosclerosis (lipoidosis) of the heart valves and its relation to endocarditis in 40 hearts with valvular disease due to atherosclerosis, endocarditis, or both. He concludes that "pure (primary) atherosclerosis (or rather lipoidosis, consists of a deposit of lipoid substances and a formation of atheromatous foci; it leads neither to the development of cicatricial tissue, nor to deformation and ulceration of the valves and, therefore, has no great independent importance in the origin of heart valvular defects. Lipoid deposition added to endocarditis in various stages of its development, presents in acute cases a picture of these two combined processes. The secondary lipoidosis (atherosclerosis) of cicatricially changed heart valves reaches a particularly high degree of development. The so-called atherosclerotic defects of the heart valves have as their basis only the atheromatous transformation of the cicatricial tissue of the valves left as a result of endocarditis. Such secondary atherosclerosis, *i.e.*, atheromatosis of the heart valves, can considerably aggravate the principal picture of a valvular defect which has arisen on the basis of a cicatricial endocarditis."

Anitschkow, N.: Atherosclerosis (lipoidosis) of the heart valves and its relation to endocarditis. Libman Anniversary Volumes. P. 651, 1932.

Aortic Valve Lesions—Campbell and Shackle have reported that in a series of cases with disease of the aortic valves, the condition was due to rheumatism in 200, to syphilis in 55, to atheroma in 20, and to all other causes in 21 cases. Out of every 6 rheumatic cases, roughly, 3 of

the patients had aortic incompetence and mitral stenosis. One had both with aortic stenosis as well, 1 had aortic stenosis and incompetence, and 1 had aortic incompetence alone. When there was no mitral disease there were 2 men for each woman; when there was mitral disease there were 2 men for every 3 women. They came under first observation at all ages, least commonly before ten or after fifty. There was so little difference between the ages of those with and those without aortic stenosis that its presence must depend on the severity and nature of the attack rather than on the length of time that had elapsed. Pure aortic stenosis was rarely found, and, so far as could be judged from the pulse pressure, the stenosis was relatively unimportant compared with the regurgitation, even when the signs were well marked. Among the syphilitic cases there were 3 men for each woman, nearly four-fifths being between forty and sixty years of age; aortic stenosis was rarely or never present, and the average pulse pressure was much greater than in the rheumatic cases (110 instead of 70). Nearly all the atheromatous cases were men, between fifty and eighty years of age; aortic stenosis was more important and more frequently present, the signs of regurgitation being often absent. Auricular fibrillation was present in 30 per cent of those who also had mitral stenosis, in 8 per cent of the rheumatic cases without mitral stenosis, and only rarely in the nonrheumatic group. Left ventricular preponderance was found in about half the electrocardiograms of those with pure aortic disease, normal limits being found in the remainder. The T waves were inverted, most often in lead I, in about one-fourth of the rheumatic cases and in about half of the others. The prognosis was enormously better in the rheumatic group; the average duration of life after the development of aortic incompetence was probably twenty years, excluding those who died quickly from active rheumatic carditis. In the syphilitic group, on the other hand, it was not much more than two years after the development of symptoms, few patients living for eight years.

Campbell, M., and Shackle, J. W.: Note on aortic valvular disease with reference to etiology and prognosis. Brit. M. J. 1:328, 1932.

Pericardial disease.—Smith and Willius have published a study of pericarditis based on 373 cases found among 8,912 necropsies at the Mayo Clinic, an incidence of 4.2 per cent. Adherent pericarditis was present in 144, or 38.4 per cent, of these 373 cases, 100 times in the male as compared with 44 times in the female sex. These patients ranged in age from two to eighty-five years. Rheumatic fever was apparently the etiological factor in 21.5 per cent; pulmonary and pleural diseases were next in frequency (17.4 per cent). Cardiac infarction was present in 6.2 per cent, neoplasm in 2.8 per cent. Only 3 cases were noted to be tuberculous. The pericardial sac was completely obliterated in 53 cases (36.8 per cent). Calcification of the pericardium was found in 15 cases (10.4 per cent). The heart weights varied greatly. Associated cardiac disease was present in only about half the cases 53.5 per cent (most commonly coronary disease) and in only 39.5 per cent were there any important cardiac symptoms or signs, due chiefly to mitral stenosis. Thus frequently asymptomatic and unimportant adherent pericarditis exists. In 87 cases of this group it was a mere incident complicating all sorts of conditions.

These authors next consider calcification of the pericardium which they found in 15 cases at necropsy; in none of these cases had the diagnosis been made during life. All 15 had extensive chronic adhesive pericarditis. Twelve were males and 3 females. The age range was from twenty-four to eighty-four years. Six cases had had rheumatic fever. Mitral stenosis was present in 3 of the cases. In 10 of the 15 cases failure of the heart was the primary cause of death.

Pericardial effusion was found by Smith and Willius in 113 cases, or 30 per cent, of all the cases of pericarditis which came to autopsy at the Mayo Clinic. Sixty-nine per cent were males and 31 per cent females. The range in age was from eight months to eighty-nine years. Acute purulent pericarditis was the most common type (68.1 per cent); fibrinous pericarditis in this series occurred in only 3 cases (2.7 per cent), while noninflammatory effusion was present also in 3 cases. The effusion varied in amount from 25 cc. to 1,500 cc. Pleural effusion was present in 73.5 per cent of the cases. Only 33 cases, or 29.2 per cent of the 113 with effusions in the pericardial sac had any associated cardiac disease, and only 13.2 per cent had any important cardiovascular symptoms.

These authors have reported finding at necropsy at the Mayo Clinic fibrinous pericarditis without effusion in 62 of 373 cases of pericarditis (16.6 per cent); the ratio of sex in this series of 62 cases was 69 per cent male to 31 per cent female. All ages were represented from four to seventy-four years. "Associated cardiac disease occurred in thirty-one cases (50 per cent). Only 20 of the 62 had any cardiovascular symptoms of importance." The condition may be innocuous in itself or it may be prodromal to more serious forms of pericarditis, with effusion, purulent or adherent. Intrathoracic infection (of lung or pleura) was apparently the cause in most of the cases (53.2 per cent). Primary pericarditis of indeterminate origin occurred next in frequency (19.4 per cent). Rheumatic fever was the cause in 14.5 per cent while infection elsewhere in the body was found in 12.9 per cent.

There were also found 15 cases with milk

spots or "soldier's patches," believed to represent relics of localized nonobliterating pericarditis; 11 of these cases were males and 4 were females.

These authors have further reported the finding of 40 cases of terminal pericarditis among 373 cases of pericarditis coming to necropsy at the Mayo Clinic, an incidence of 10.7 per cent. Of these 40 patients, 28 were males and 12 females. The age limits were fifteen and eighty-three years. Heart disease was often associated, but the heart was predominantly a factor in the clinical syndrome in only 4 cases. Coronary disease occurred in only 3 cases and rheumatic heart disease also in only 3. Bacterial endocarditis was present in one case. The major diagnosis in 32.5 per cent was carcinoma and in 25 per cent nephritis (with uremia). Infections occurred in only half the cases.

Camp and White have presented clinical and pathologic data on 126 cases containing over 100 cc. of pericardial fluid found at postmortem examination at the Massachusetts General Hospital over a period of ten years (1921 to 1930 inclusive), similar data on two cases at the Massachusetts Eye and Ear Infirmary, and clinical data on 15 other cases in which pericardial paracentesis established the diagnosis of pericardial effusion. "The predominant etiologic factor in the cases with pericarditis was infection of some type (uremia was a rare cause). In cases without pericarditis the predominant etiologic factor was chronic passive congestion or infection elsewhere in the body. . . . Pain was present in 24 of the 126 cases of the postmortem series of the Massachusetts General Hospital, dyspnea in 68, orthopnea in 37, distended cervical veins were noted in 5, pericardial friction rub in 8, distant heart sounds in 29, and the pulsus paradoxus in 1 case. Forty-nine cases were examined by roentgen ray. A correct diagnosis was made once and questioned in 3 other cases. Allowances must be made because many of these cases were examined at least several days before death and often in a very ill condition, with pleural fluid sometimes obscuring a part of the heart shadow. Of the 126 cases of the postmortem series at the Massachusetts General Hospital a correct clinical diagnosis was made only six times, in 4 of the 94 cases with between 100 and 250 cc. of pericardial fluid, in none of the 24 cases with 250 to 500 cc. of fluid, and in 2 of the 8 cases with over 500 cc. of fluid." "The clinical group of 15 other cases in whom a pericardial paracentesis established the diagnosis of pericardial effusion was most instructive since it was made up of patients who presented the classical signs of effusion in a much greater degree than did the 'postmortem' cases." All but one of these clinical cases undoubtedly had over 500 cc. of fluid in the pericardium; the one who had considerably less showed at operation a much thickened contracted parietal pericardium. The authors conclude that "without the presence of an acute fibrinous pericarditis, the diagnosis of pericardial fluid is likely to be missed unless the effusion amounts to over 500 cc."

Foulger and Foulger have reported experiments on dogs showing that a fall in arterial blood pressure accompanies an increase in intrapericardial pressure, as does also decreased voltage of the QRS wave of the electrocardiogram with inversion of the T wave which may finally resemble a coronary T wave. "An intrapericardial pressure of 3 to 8 mm. Hg. is sufficient to produce an obviously negative T-wave" in these experimental animals.

Sprague, Burch and White have reported a study of the autopsy material at the Massachusetts General Hospital from 1921 to 1931 for cases of adherent pericardium (43 cases, or 2.26 per cent of 1900 cases) and from 1893 to 1931 for cases of Pick's disease or mediastinopericarditic pseudocirrhosis of the liver (only 3 cases in this period of 38 years). No case of rheumatic pericarditis either in the Massachusetts General Hospital series or in a clinical group of 62 patients at the House of the Good Samaritan has ever developed Pick's disease. The authors conclude that it is unjustifiable to recommend pericardial resection following acute rheumatic pericarditis to prevent later effects of adhesions, also that tuberculosis of an insidious type is the most probable cause of the majority of cases of Pick's disease, and that, in middle and old age, adhesive pericarditis with or without calcification is an unimportant cause of symptoms and death except in very rare instances.

Camp, P. D., and White, P. D.: Pericardial effusion; a clinical study. Am. J. M. Sc. 184:782, 1932.

Foulger, M. and Foulger, J. H.: The blood pressure and electrocardiogram in experimental pericardial effusion. Am. Heart J. 7:744, 1932.

Smith, H. L., and Willius, F. A.: Pericarditis. I. Chronic adherent pericarditis. II. Calcification of pericardium. III. Pericarditis with effusion. Arch. Int. Med. 50:171, 184, 192, 1932.

IV. Fibrinous pericarditis and "soldier's patches". Arch. Int. Med. 50:410, 1932.

V. Terminal pericarditis. Arch. Int. Med. 50:415, 1932.

Sprague, H. B.; Burch, H. A., and White, P. D.: Adherent pericardium and Pick's syndrome. An autopsy study. New. Eng. J. Med. 207:483, 1932.

Vascular Disease (Including Lesions of Aorta and Pulmonary Artery):

Aortic Pathology.—Klotz and Simpson have studied 5 cases of so-called spontaneous rupture of the aorta. "A common underlying process was demonstrated in all cases, consisting of a peculiar noninflammatory degeneration of the media, affecting the muscle and elastic fibers, due to a variety of factors. Similar lesions precede the development of dissecting aneurysms. The lesions have no relation to

syphilis." The 5 cases were aged twenty-three, forty-five, fifty-four, thirty-nine, and thirty-eight years old respectively. There were 3 females and 2 males in the series.

Wood, Pendergrass, and Ostrum have reviewed the subject of dissecting aortic aneurysm with especial reference to its roentgenographic features. They report 11 new cases, 5 of their own and 6 from the literature to add to the group of about 400 previous cases reported. Of this earlier group only 6 had been correctly diagnosed clinically; there is no note that any were so diagnosed in the later group.

Klotz, O., and Simpson, W.: Spontaneous rupture of the aorta. Am. J. M. Sc. 184:455, 1932.
Wood, F. C.; Pendergrass, E. P., and Ostrum, H. W.: Dissecting aneurysm of aorta, with special reference to its roentgenographic features. Am. J. Roentgenol. 28:437, 1932.

Pulmonary Arterial Disease.—Gibbon, Hopkinson, and Churchill have determined that obstruction of the pulmonary artery up to 60 per cent of its cross sectional area in cats is without significant effect upon either the arterial or the venous pressure. When the occlusion lies between 60 and 85 per cent there is a fall in blood pressure and a rise in venous pressure. "The circulation fails not primarily from cardiac insufficiency but due to the fact that blood collects on the venous side of the system by reason of the obstruction to the outflow from the right heart. The obstruction is fatal when 85 to 100 per cent of the pulmonary artery is occluded."

Gibbon, J. H., Jr.; Hopkinson, M., and Churchill, E. D.: Changes in the circulation produced by gradual occlusion of the pulmonary artery. J. Clin. Investigation. 11:543, 1932.

General Arterial Disease.—Waters has discussed the treatment of peripheral vascular diseases with acetylcholine hydrochloride (50 to 100 mg. subcutaneously or intramuscularly, to be repeated at twelve to twenty-four hour intervals; administered orally, it is ineffective and when given intravenously, it is very toxic and may kill). He reports healing of gangrenous areas, relief of pain, and elevation of surface temperature in 3 patients with trophic lesions due to vascular disease of the extremities. This drug has the advantage over typhoid vaccine in that there are no constitutional reactions following its use.

Winkelman and Eckel have written that lesions of the spinal cord can result from vascular disease, such as arteriosclerosis, thrombosis, rupture, or emboli. The lesions may be small and scattered or may involve a complete transverse level. The scattered small lesions may resemble multiple sclerosis. The transverse softening may resemble a compression syndrome. Syphilis plays an important rôle in vascular disease. Unusual conditions, such as angioma and carcinoma, must be kept in mind in the diagnosis of lesions of the spinal cord.

Waters, W. C., Jr.: Peripheral vascular diseases. Treatment with acetyl-choline hydrochloride. Ann. Int. Med. 5:1267, 1932.
Winkelman, N. W., and Eckel, J. L.: Focal Lesions of the Spinal Cord Due to Vascular Disease. J. A. M. A. 99: 1919, 1932.

Diseases of the Veins.—Homans has published a good review of the subject of thrombophlebitis. In summary he has said, "There are forms of thrombophlebitis dependent upon an unhealthy state of the vein's wall, the varicose type, and peculiar sorts, partly of local origin and partly due, perhaps, to an abnormality of the blood—phlebitis migrans. There are also venous thromboses of traumatic origin. But thrombophlebitis which affects the previously healthy veins draining the lower limbs, particularly the familiar scourge, phlegmasia alba dolens, appears to be secondary to a nonsuppurative lymphangitis, which from its situation, is able to attack artery as well as vein. And the principal exhibitions of such a disease are neither venous nor arterial, but lymphatic."

Moschcowitz has described for the first time sclerosis of the hepatic veins associated with hypertension of the pulmonary circuit. He cites 3 cases all discovered within a few weeks, and believes the lesion to be very common. He states that "sclerosis of the hepatic veins is only present when a condition causing increased pressure in the hepatic veins can be predicated, the result in every instance of hypertension of the pulmonary circuit with right heart failure. It is always associated with passive venous congestion of the liver with and without cardiac cirrhosis. The lesion consists in dilatation of the hepatic veins with thickening of both intima and media and proliferation of the elastic fibres, and is interpreted as compensatory to the increased intravenous pressure."

Homans, J.: Phlegmasia alba dolens and the relation of the lymphatics to thrombophlebitis. Am. Heart J. 7:415, 1932.
Moschcowitz, E.: Phlebosclerosis of the Hepatic Veins. Libman Anniversary Volumes. 2:857, 1932.

Aneurysms:

Aortic—Aschner has reported a case of gonococcus mycotic aneurysms of the ascending aorta, four in number, in a woman twenty-three years old. He concludes with a review of the literature on mycotic aneurysms.

White has reported successful relief of pain caused by aneurysms of the aortic arch in 3 cases by paravertebral injections of alcohol (2 cases) and procaine alone (1 case). He writes: "Procaine injection is of diagnostic value in determining which communicant rami transmit the painful impulses to the spinal cord. It may give relief over prolonged periods. Alcohol, when accurately injected, causes permanent destruction of the sympathetic rami. Sympathetic

ganglionectomy although a surer method of interrupting cardio-aortic pain pathways, is too serious an operative procedure to use in patients with large thoracic aneurysms. Paravertebral block, although technically a difficult procedure, is well tolerated and should give satisfactory results in a large percentage of cases."

Aschner, P. W.: Gonococcus aneurisms of the aorta. Libman Anniversary Volumes. P. 75, 1932.
White, J. C.: Painful aneurysms of the aortic arch; relief by paravertebral injections of procaine and alcohol. J. A. M. A. 99:10, 1932.

ARTERIOVENOUS — Matas, Herrmann and Reid have reported a case showing the effect of an arteriovenous aneurysm on the heart. The aneurysm was femoral and had been present for seventeen years. There were cardiac hypertrophy and dilatation. Excision of the aneurysm relieved completely all cardiac symptoms and caused the heart to return to normal size.

Horton has discussed a study made of 23 cases out of the 24 who were diagnosed congenital arteriovenous fistula at the Mayo Clinic from June 28, 1929, to May 11, 1931. Eleven were males, 12 females. The right arm was involved in 3 cases, the left arm in 5, the right leg in 4, the left leg in 11. In only one case was a correct diagnosis made before entry to the Clinic. Most of the others were diagnosed varicose veins. Cardiac hypertrophy was definite in 8 cases. Hypertension was found in 6 cases, tachycardia (over 80) in 18 cases. The affected limb was hypertrophied in each case.

Horton, B. T.: Hemihypertrophy of extremities associated with congenital arteriovenous fistula. J. A. M. A. 98:373, 1932.
Matas, R.; Herrmann, G., and Reid, M. R.: Effect of arteriovenous aneurysms of heart: report of case studied. Ann. Surg. 95:578, 1932.

FUNCTIONAL DISORDERS

Congestive Failure, Including the Use of Digitalis and Diuretics.

Austrian has reported an interesting case of encapsulated hydrothorax on the right side in association with congestive heart failure in a man fifty-eight years old. Postmortem examination showed syphilitic aortitis, aortic insufficiency, obliteration of the orifice of the right coronary artery, cardiac hypertrophy and dilatation, and pleural adhesions.

Harrison and his associates have studied the mechanism of dyspnea on exertion in cardiac patients and have arrived at the following conclusions: (1) A decrease in vital capacity is important in two respects: (a) *Per se,* it lowers the respiratory reserve and thereby predisposes to dyspnea, (b) It increases the resting ventilation through vagal reflexes from the lungs and hence lowers the respiratory reserve still further. (2) Afferent impulses from the moving muscles play a rôle in the production of dyspnea because they cause reflex increase of the ventilation during the exertion. (3) Reflex stimulation of respiration, because of increased pressure in the right side of the heart and in the cardiac ends of the great veins, is of especial importance. (4) All these factors so operate as to increase the value of the quotient $\frac{\text{ventilation}}{\text{vital capacity}}$, which is a measure of subjective respiratory distress in persons with cardiac disease.

Meakins has written that "There are three important known conditions which may lead to failure of the cardiac muscle to function properly through a defect in cardiac glycogen metabolism. They are oxygen want, thyrotoxicosis, and insulin deficiency."

Austrian, C. R.: Encapsulated hydrothorax (hydrothorax saccatus interlobaris) in association with myocardial insufficiency. Libman Anniversary Volumes. P. 101, 1932.
Harrison, T. R.; Harrison, W. G.; Calhoun, J. A., and Marsh, J. P.: Congestive heart failure. XVII. The mechanism of dyspnea on exertion. Arch. Int. Med. 50:690, 1932.
Meakins, J. C.: Modern muscle physiology and circulatory failure. Ann. Int. Med. 6:506, 1932.

Digitalis Action—Cohn and Steele have found that "the minute output from failing, dilated hearts in dogs in heart lung preparations is increased when therapeutic doses of digitalis are administered. This effect is the opposite of that in the case of healthy hearts, normal in size. When the output increases, the pressure in the right auricle decreases. Increase in output is consistent with decrease in the diastolic volume of the heart."

Stewart and Cohn have found that in dogs "when the cardiac output is diminished and the heart is dilated due to artificial auricular fibrillation, the administration of digitalis results in increase in cardiac output and decrease in cardiac size." These observers concluded that "digitalis has the same action in normal and in pathological hearts; it decreases cardiac size (an effect on tone). The amount of the cardiac output which results from this action depends upon the initial size of the heart; it decreases in normal hearts, and increases in dilated ones."

Stewart and Cohn have continued their studies of the effect of digitalis on heart size and output, this time in man and have concluded as follows:

"(1) A consequence of the action of digitalis is to decrease the volume output of blood per minute from the heart in normal human beings and to decrease its size. (2) The volume output of blood per minute from the heart which is in failure is diminished and its size larger than when it is in a state of compensation. (3) Following the administration of theocalcin in patients during heart failure, cardiac output increases and cardiac size and venous pressure diminish. (4) Giving digitalis increases the volume output of blood per minute from failing hearts and decreases their size. (5) Digitalis, we think, has similar, perhaps identical, actions both in normal and in diseased hearts; it *de-*

creases cardiac size and *increases* the extent of ventricular contraction. The consequence of these actions is that the volume of the cardiac output which results differs, depending on an initial difference in size of the ventricular cavities, in the two situations. In the one, the normal heart, it becomes too small, in the other, the diseased heart, it develops a suitable size."

Cohn, A. E., and Steele, J. M.: Studies on the effect of the action of digitalis on the output of blood from the heart. I. The effect on the output of the dog's heart in heart-lung preparations. J. Clin. Investigation. 11:871, 1932.

Stewart, H. J., and Cohn, A. E.: Studies on the effect of the action of digitalis on the output of blood from the heart. II. The effect on the output of the hearts of dogs subject to artificial auricular fibrillation. J. Clin. Investigation. 11:897, 1932.

Studies on the effect of the action of digitalis on the output of blood from the heart. III. Part 1, The effect on the output in normal human hearts; Part 2, The effect on the output of hearts in heart failure with congestion in human beings. J. Clin. Investigation. 11:917, 1932.

Diuretics—Herrmann and his associates have made careful studies of the action of diuretic drugs on 12 cases of congestive heart failure with edema, using particularly among other tests the creatinine method of Rehberg, in order to try to determine how these diuretics work. Among their conclusions are the following:

"Rest, digitalis and theophylline-ethylene-diamine (euphylline) apparently result in augmented glomerular tuft and tubular capillary circulation, with a consequent increased glomerular filtration and a concomitant slightly impaired or less efficient tubular reabsorption to account for the increased urinary output.

"Mersalyl, or salyrgan, seems to produce diuresis primarily by depressing the cells of the tubular epithelium, thereby hampering diffusion or reabsorption, while filtration is little if at all affected. The defective reabsorption is great enough to account for the diuresis. . . .

"From our studies it appears that the action of diuretics is primarily on the kidney and only secondarily on the tissue fluids."

Miller and Feldman have reported on the prolonged use of massive doses of urea in cardiac dropsy, giving 5 detailed case reports. The doses of urea varied between 10 and 25 grams, two or three times a day in 40 per cent water solution. Whenever possible, at the beginning of treatment, the patients received about 18 grams three times a day (rarely 27 grams three times daily). After satisfactory diuresis the drug was gradually reduced, the patients receiving 15 or even 12 grams three times or twice daily for months to prevent the reaccumulation of edema. As a general rule, the patients took the drug without remonstrance although its taste is very unpleasant. Sometimes the addition of syrups helped. The authors conclude that "urea administered in the way described has been found to be an effective diuretic over long periods of time in selected cases. Whereas it can function alone over many months in keeping the patient edema-free, it may require the supplemental use of other diuretics. This is true for the patient with or without auricular fibrillation. Cases are reported in which its unbroken administration continued over three years. Urea does not impair the function of the kidneys or alter their histologic structure after prolonged administration. . . . The drug tends to maintain the patient's weight at a low and constant edema-free level over months and even years. During such periods, our patients often required little or no restrictions of the intake of fluid or salt within ordinary dietary limits."

Schmitz has conducted experiments on dogs to compare the diuretic action of euphyllin and salyrgan by Rehberg's method. He has found that "during euphyllin diuresis the calculated amount of glomerular filtration is consistently increased while tubular reabsorption shows no constant change. Salyrgan diuresis is characterized," he found, on the other hand, "by a considerable decrease in the percentage of fluid reabsorbed in the tubules. The amount of filtration is seldom increased significantly."

Tarr and Jacobson have reviewed the comparative toxicity of salyrgan and novasurol and have noted the inconsistency between the clinical findings and the results of animal experimentation. "In more than 3,000 injections of salyrgan given at the Montefiore Hospital only two instances of mild stomatitis were encountered when the drug was administered to patients with impaired renal function. In one patient wrist-drop developed and in two foot-drop following faulty intramuscular injection. Anatomic studies were made of thirty patients with congestive heart failure who were given repeated injections with doses of salyrgan up to 130 cc. In one case a lesion suggestive of mercurialism was found at autopsy in the kidney."

Wiseman has reported the successful use of novasurol and salyrgan in 270 injections over a period of five years in the case of an obese woman with hypertensive cardiovascular disease and congestive failure. "An excellent diuretic response was maintained throughout. The prolonged diuretic treatment appeared to be without harmful effects on the kidneys or other organs."

Stockton has reported his finding that "the intramuscular injection of bismuth sodium tartrate into fifteen patients was followed by a definite and sustained increase in diuresis in 73 per cent." The dose injected was 0.03 gram of the bismuth compound.

Herrmann, G.; Stone, C. T.; Schwab, E. H., and Bondurant, W. W.: Diuresis in patients with congestive heart failure. J. A. M. A. 99:1647, 1932.

Miller, H. R., and Feldman, A.: Prolonged use of massive doses of urea in cardiac dropsy. Arch. Int. Med. 49:964, 1932.

Schmitz, H. L.: Studies on the action of diuretics. I. The effect of euphyllin and salyrgan upon glomerular filtration and tubular reabsorption. J. Clin. Investigation. 11:1075, 1932.

Stockton, A. B.: Bismuth diuresis and blood and urinary changes under clinical conditions. Arch. Int. Med. 50: 142, 1932.

Tarr, L., and Jacobson, S.: Toxicity of mersalyl (salyrgan): clinical and anatomic study. Arch. Int. Med. 50:158, 1932.

Wiseman, J. R.: The prolonged use of salyrgan. J. A. M. A. 99:114, 1932.

Functional Vascular Disorders.

Erythromelalgia has found fundamental criteria, according to Brown: (1) bilateral burning pain in the extremities, (2) sharp increase in local heat of the affected parts, (3) production and aggravation of the distress by heat and exercise, and (4) relief by rest, cold, and elevation. Its etiology is unknown. Vasodilatation in the presence of polycythemia vera is common.

Allen and Brown have published an analysis of 204 cases of Raynaud's disease observed at the Mayo Clinic between January 1920 and July 1931. These cases they grouped as follows: uncomplicated, 147 cases; with scleroderma and arthritis and with or without gangrene or recurrent infections, 51 cases, and with gangrene or recurrent infections unassociated with scleroderma or arthritis, 6 cases. "Study of 147 cases of Raynaud's disease of the uncomplicated type indicates the disease is ordinarily an equivalent of psychoneurosis or neurasthenia. Operation on the sympathetic nervous system is the best method of treatment, offering a cure in the uncomplicated type of case."

White has published an interesting report of 6 cases of typical Raynaud's disease, with observations on the immediate and late effects of sympathetic neurectomy.

"Resection of the two upper dorsal ganglia or of the second to fourth lumbar ganglia brought about an immediate paralysis of sympathetic tonus in each case. Whereas vasomotor paralysis following lumbar sympathectomy has been permanent, the dorsal operation has been followed by a recurrence of sympathetic nerve function in 2 out of the 5 cases reported here and of 5 more operated on outside the hospital by members of the Vascular Clinic. Sympathetic nerve activity has reappeared at the end of six months, accompanied by the color changes, pain, coldness, and ulceration in the tips of the fingers characteristic of Raynaud's disease. When the regenerated vasoconstrictor fibres were again adequately blocked by novocaine or by re-operation, there was a second disappearance of the manifestations of the disease. Therefore, resection of the first and second dorsal sympathetic ganglia alone is insufficient to cause a permanent vasomotor paralysis of the arm. In our recent cases, where the operation has been extended upwards to include the inferior cervical ganglion, we are hopeful that the results will be as permanent as those in the lower extremity.

"Advanced stages of the disease with long-standing ulceration and sclerodermatous changes may fail to recover completely after vasomotor paralysis. These instances are satisfactorily explained by Lewis' theory of local pathology in the digital vessels.

"From the theoretical standpoint, early uncomplicated cases of the disease may be explained either on the basis of Raynaud's original conception of a vasomotor neurosis or by Lewis' theory that normal vasoconstrictor impulses cause the attacks of partial asphyxia by acting on abnormal digital arterioles. Certain objections have been pointed out against each hypothesis. It is therefore essential to reserve final judgment until the evidence in favor of one or of the other becomes unequivocal."

Allen, E. V., and Brown, G. E.: Raynaud's disease. J. A. M. A. 99:1472, 1932.
Brown, G. E.: Vasodilation disturbances affecting the extremities: A clinical study. Libman Anniversary Volumes. P. 241, 1932.
White, J. C.: Raynaud's disease. Studies on post-operative cases bearing on the etiology of the disease and the efficiency of sympathetic ganglionectomy. New Eng. J. Med. 207:1198, 1932.

Angina Pectoris.— Lian and his associates have published during the past year an extensive discussion of angina pectoris and other kinds of "heart pain" in the form of a volume of 425 pages, the first half of which takes up in detail the anginal syndrome itself and acute coronary thrombosis, while the second half takes up heart pain of other types, thoracic distress of reflex, neurotic, and toxic origin, and the differential diagnosis, prognosis, and treatment of all kinds of substernal and precordial pain. More attention than usual is directed to reflex pain and to medicinal and other types of nonsurgical treatment. The title does not indicate sufficiently the wide scope of the discussion.

Katz, Hamburger, and Lev studied the effect of hypodermic injections of 1 cc. of 1:1,000 solution of epinephrine on a group of 6 cases of known angina pectoris, on a group of 6 normal internes, on a case of "irritable heart" with precordial distress, and on a case of luetic aortitis with precordial distress. The authors found epinephrine as a test for angina pectoris "to be unreliable in regard to both the symptoms produced and the electrocardiographic changes. This unreliability makes the test of little value, particularly in view of the severe reactions sometimes encountered." Three of the 6 cases of angina pectoris had no pain of any sort resulting from the injection of epinephrine and the others had pain not similar to their angina pectoris, except perhaps in one case.

White and Camp have reported 4 cases of status anginosus induced by tachycardia, in 3 cases paroxysmal auricular fibrillation and in the fourth case paroxysmal tachycardia. All 4 patients showed angina pectoris on effort. Two were men aged sixty-two and sixty-six years, respectively, and two were women aged sixty-eight and seventy-five. One of the men and one of the women died suddenly eighteen months and fifteen months, respectively, after the first

attack of angina pectoris induced by the abnormal heart rhythm.

Nuzum and Elliot have reported the results of their administration intramuscularly of an insulin-free extract of the pancreas to 20 patients with angina pectoris and to 5 with intermittent claudication. This extract is a vasodilator that modifies the pressor effects of epinephrine and dilates the coronary arteries of the rabbit's heart to a degree exceeding that produced by drugs of the purine group. Of the cases of angina pectoris, "two, or 10 per cent, were not helped; five, or 25 per cent were somewhat relieved; eleven, or 55 per cent, were greatly helped, and two who were benefited by treatment died later. Forty-one patients with angina pectoris who received the usual medical treatment and were followed for a like period of time were studied as a control series. In ten instances, or 24.3 per cent, no benefit was observed; fourteen patients, or 34.1 per cent, experienced moderate relief; fourteen, or 34.1 per cent, received pronounced benefit, and three, or 7.4 per cent, died." The cases of intermittent claudication were all benefited to a pronounced degree. The extract was given intramuscularly in doses of from 30 to 60 hypotensive units on alternate days or twice weekly, depending on the severity of the symptoms, for a total of from ten to twelve doses. Two or three such courses were given to some of the cases at intervals of a week.

Katz, L. N.; Hamburger, W. W., and Lev, M.: The diagnostic value of epinephrine in angina pectoris. Am. Heart. J. 7:371, 1932.
Lian, C., and associates: L'Angine de Poitrine. Formes Cliniques, Traitement Medical et Chirurgical. 425 pages. 34 figures. Masson et Cie., Paris, 1932.
Nuzum, F. R., and Elliot, A. H.: Pancreatic extract in treatment of angina pectoris and intermittent claudication. Arch. Int. Med. 49:1007, 1932.
White, P. D., and Camp, P. D.: The status anginosus induced by paroxysmal auricular fibrillation and paroxysmal tachycardia. Am. Heart J. 7:581, 1932.

Arrhythmias: Premature Beats and Tachycardia.—Sampson and Anderson have published an interesting report of the results of their administration of potassium salts orally to 58 patients who were known to have auricular or ventricular ectopic beats or paroxysmal auricular or ventricular tachycardia. "One of the patients received in addition 10 cc. of potassium chloride intravenously in a 1 per cent solution without ill effect, but with no demonstrable advantage over the oral route of administration. 'Five' different salts of potassium were used, namely, the chloride, iodide, bromide, citrate, and acetate, all with apparently identical effects when applied to the same patient (Case 3). As a control measure, sodium acetate was given and produced no change in the arrhythmia. . . . The acetate caused less gastric distress than any of the other salts, so this compound was adopted for routine use, in a 25 per cent aqueous solution." The effective dosage varied from 2 to 16 grams. Response occurred in the majority of cases in about forty minutes, ranging from twenty to ninety minutes. The duration of effect ranged from three to eight hours. An effective dose has been found to be 2 to 4 grams every six to eight hours and may be given over long periods of time with effect and without danger (in one case two years and two months).

In the 58 cases of arrhythmia studied, complete relief was obtained in 29; 6 others showed some benefit, and 3 apparently were made worse. Toxic symptoms were rare; they were gastric distress, abdominal cramps, and diarrhea; diuresis was uncommon.

Capo reports, and illustrates with several electrocardiographic records, four observations of bigeminal rhythm occurring in heart patients with grave signs of cardiac insufficiency, following administration of small doses of digitalis or of ouabain. He analyzes the various pathogenic theories of this disturbance of cardiac rhythm and states that the appearance of bigeminal rhythm in the course of cardiac insufficiency may be considered a sign of grave prognosis. The patients studied died within two or three months after the development of the bigeminal rhythm.

Anderson has reported an interesting case of a woman twenty-two years old with paroxysmal tachycardia, who was entirely free from paroxysms throughout her pregnancy but had attacks during the last menstrual period before the pregnancy and the first one after delivery.

Anderson, D. F.: Paroxysmal tachycardia associated with pregnancy. Brit. M. J. 1:657, 1932.
Capo, R.: Regular extrasystolic bigeminal rhythm associated with cardiac insufficiency. Clin. med. ital. 62: 627, 1931.
Sampson, J. J., and Anderson, E. M.: The treatment of certain cardiac arrhythmias with potassium salts. J. A. M. A. 99:2257, 1932.

Auricular Fibrillation and Auricular Flutter.

McEachern and Baker have reported a study of 575 consecutive cases of auricular fibrillation confirmed by electrocardiogram. The chief etiological relationships were as follows: rheumatic heart disease, 34.4 per cent, coronary disease and old age, 31.1 per cent, hypertension 16.9 per cent, thyrotoxicosis, 7.5 per cent, emphysema 5.0 per cent, syphilis 3.0 per cent, miscellaneous, 2.1 per cent. The age incidence was as follows:

0- 9 = 0.5 per cent	50-59 = 26.1 per cent
10-19 = 4.2 per cent	60-69 = 20.5 per cent
20-29 = 5.4 per cent	70-79 = 7.8 per cent
30-39 = 15.0 per cent	80-89 = 0.3 per cent
40-49 = 20.2 per cent	

Stroud, Laplace, and Reisinger have reported their study of the etiology, prognosis, and treatment of 253 cases of chronic auricular fibrillation. They found no common etiologic factors

and concluded that "the presence of auricular fibrillation does not necessarily imply a worse prognosis than in the unfibrillating heart with an equal amount of cardiovascular damage."

In this series, the average age of onset of auricular fibrillation due to rheumatic cardiovascular damage was thirty-nine years. The prognosis was very poor in patients developing this arrhythmia before the age of twenty-five years. The average age of onset of auricular fibrillation in the arteriosclerotic (nonrheumatic) group was about fifty-nine years.

As to treatment of patients with auricular fibrillation, the authors conclude that "digitalis usually proves most satisfactory." "Although there appears to be little danger of accident from the use of quinidine sulphate in properly selected cases of auricular fibrillation, yet we feel this drug should only be used in those few cases of younger individuals with slight or no demonstrable cardiovascular abnormality except the arrhythmia, or in thyrotoxic patients following partial thyroidectomy without a spontaneous return to normal sinus rhythm."

McMillan and Bellet have published an analysis of 65 cases of auricular flutter; among 55 of these 12 were paroxysmal and 43 established; 7 of the remaining 10 were due to drugs (including digitalis) and 3 were unclassified. Normal rhythm was restored in two-thirds of the cases. The method of choice was digitalis therapy until auricular fibrillation was established and then quinidine if normal rhythm did not occur spontaneously.

McEachern, D., and Baker, B. M., Jr.: Auricular fibrillation. Its etiology, age incidence and production by digitalis therapy. Am. J. M. Sc. 183:35, 1932.
McMillan, T. M., and Bellet, S.: Auricular flutter; some of its manifestations and its treatment; based on study of sixty-five cases. Am. J. M. Sc. 184:33, 1932.
Stroud, W. D.; Laplace, L. B., and Reisinger, J. A.: The etiology, prognosis and treatment of auricular fibrillation. Am. J. M. Sc. 183:48, 1932

Quinidine therapy—Levine has reported a series of 36 experiments on cats demonstrating that quinidine sulphate tends to inhibit the occurrence of ventricular fibrillation on faradic stimulation.

Newman and Spiro have reported their experience with the use of quinidine in treating auricular fibrillation in a series of 66 patients over an interval of several years. Sinus rhythm was restored in 54 cases, or 82 per cent. Of these 54 patients, 12, or 30 per cent, reverted to auricular fibrillation before the end of one year, 28, or 70 per cent, maintained normal rhythm for one year or more, 28, or 57 per cent, for two years or more, 14, or 35 per cent for three years or more, 8, or 20 per cent, for four years or more, 3, or 7 per cent, for five years or more, and 2, or 5 per cent, for six years or more. Of the 66 patients who were treated, 3 died within a few hours of the restoration of normal rhythm; 3 more patients were alarmingly poisoned and 6 moderately so. The larger the heart, the less satisfactory the therapy.

McGuire has treated "approximately 50" patients with "extrasystolic irregularities" with quinidine sulphate (2 to 5 grains, three times a day) in the Cincinnati General Hospital over an interval of several months and has found that in a small group of the cases without evidence of heart disease the drug abolished the extrasystoles or reduced them in frequency in 85 per cent. In the second group, patients with organic heart disease but without congestive failure, the drug rarely caused the premature beats to disappear; it did, however, usually diminish their frequency.

Levine, H. D.: Effect of quinidine sulphate in inhibiting ventricular fibrillation; experimental study. Arch. Int. Med. 48:808, 1932.
McGuire, J.: Extra-systoles and their treatment with quinidine sulphate. Ohio State M. J. 28:260, 1932.
Newman, W. W., and Spiro, H.: Quinidine therapy in auricular fibrillation. California & West. Med. 37:19, 1932.

Heart Block: *Auriculoventricular.*—Mahaim has written an extensive monograph on the pathology and clinical manifestations of lesions of the bundle of His and its branches, with detailed reports of the histological examination of serial sections in 20 cases, the majority showing either A-V block, bundle branch block, or both. A notable conclusion is that rarely is there a localized lesion in the A-V conduction system; main bundle and both branches are usually all affected in greater or lesser degree in the same case.

Don, Grant and Camp have reported a case of complete heart block in which the ventricular complexes of the electrocardiogram varied in form, and which showed at postmortem examination destruction of the bifurcation of the A-V bundle, thus separating the main bundle from its branches and the right branch from the left. The patient was a man aged sixty-eight years.

Grant and Camp have described a case of complete heart block developing in an adult (a man aged forty years) during the last few months of life. At postmortem examination the terminal portion and bifurcation of the A-V bundle were found destroyed by an arterial angioma.

Don, C. S. D.; Grant, R. T., and Camp, P. D.: A case of complete heart block with varying ventricular complexes. Heart. 16:145, 1932.
Grant, R. T., and Camp, P. D.: A case of complete heart block due to an arterial angioma. Heart. 16:137, 1932.
Mahaim, I.: Les Maladies Organiques du Faisceau de His-Tawara. Les Syndromes Coronaires. L'Endocardite Septale. L'Infarctus Septal. Étude Clinique et anatomique. Masson et Cie., Paris, 1931. 595 pages, 188 illustrations.

Effect of Epinephrine—Schwartz and Jezer have reported Adams-Stokes attacks in 4 cases, 2 with ventricular standstill at intervals and 2 with periods of ventricular fibrillation, both mechanisms causing syncope. Adrenalin is in-

dicated in the case of ventricular standstill and contraindicated in the case of ventricular fibrillation.

Blumgart has reported the effect of the subcutaneous injection of 1/2 to 1 cc. of 1:1000 solution of epinephrine on the circulation in 10 subjects. The velocity of blood flow through the lungs was increased in every subject, on the average of 76 per cent. "The velocity of blood flow from the arm to the heart, the pulse rate and the systemic arterial blood pressure were likewise increased. The metabolic rate was elevated by the administration of epinephrine, the increase ranging from 8 to 22 per cent and averaging 16 per cent."

Blumgart, H. L.: The circulatory response to epinephrine. Libman Anniversary Volumes. P. 215, 1932.
Schwartz, S. P., and Jezer, A.: The action of adrenalin on patients with complete heart-block and Stokes-Adams seizures. Am. Heart J. 7:652, 1932.

Intraventricular. — Wilson, Macleod, and Barker have published data of their experimental work on dogs and clinical observations on 6 patients with bundle branch block which demonstrate conclusively that the old nomenclature must be revised, the common type of bundle branch block being left branch block, the rare type right branch block. They made their observations chiefly by the use of semidirect leads (one electrode, the exploring electrode being placed close to the heart, the other—the indifferent electrode—at a distance from the heart, that is on a leg). Semidirect leads are essentially similar to direct leads. "In animals in which bundle branch block has been produced, the chief upstroke of the ventricular complex occurred early in those semidirect leads in which the exploring electrode was close to the surface of the contralateral, and late in those leads in which the electrode was close to the surface of the homolateral ventricle." "Serial precordial leads in cases of clinical bundle-branch block of the common type yield results similar to those obtained in experimental right branch block."

Smith reports that of 47 cases with bundle branch block, 44 died within eighteen months after coming under observation.

Wilson, F. N.; Macleod, A. G., and Barker, P. S.: The order of ventricular excitation in human bundle-branch block. Am. Heart J. 7:305, 1932.
Smith, A. L.: One sided branch block: A review of forty-seven cases. Nebraska M. J. 17:179, 1932.

WHERE THE TYPHOID FEVER DEATHRATE IS STILL HIGH

As measures for the control of typhoid fever have been more and more widely extended, the disease, from a country-wide standpoint, has become a minor factor as a cause of death. This is clearly shown by the fact that, in 1932, the deathrate in seven states was less than 1 per 100,000, and in twenty states less than 2 per 100,000.

But, effective as has been the progress of sanitary science and of civic and domestic hygiene in reducing the typhoid fever death toll, current records of mortality show that we are still far from the goal of absolute control.

There were, in 1932, seven states which still had typhoid fever deathrates of over 10 per 100,000. They comprise only 13 per cent of the total population of the country, but they contributed 45 per cent of all the deaths from typhoid. Furthermore, there were nine more states in which the 1932 typhoid fever rate ran 5 or over per 100,000. In the area within these sixteen states, containing only 29 per cent of the population, 71 per cent of all the typhoid fever deaths occurred last year.

The states with typhoid fever deathrates in excess of 10 per 100,000, in 1932, were South Carolina (14.6), Kentucky (13.0), Georgia (12.5), Arkansas and West Virginia (each 12.4), Tennessee (11.6), and Louisiana (10.9). The average typhoid deathrate in this group is today about double the average rate for the entire Death Registration Area ten years ago. These are all Southern or "Border" states; and one might surmise that their high rates were due, solely, to excessive mortality from this disease among their Negro populations. But this is by no means the case. In all of these states the whites suffer high typhoid mortalities, running from nearly double to more than three times the average for the white population of Continental United States. The highest figure is that of Kentucky (12.7 per 100,000), followed by Arkansas, with 10.4; Tennessee, 9.8; Louisiana, 7.8; Georgia, 7.2 and South Carolina, 5.8. The figures for West Virginia, by color, are not available at this writing.

Although it is true that, for the most part, typhoid control is a problem of rural sanitation, high mortality rates are by no means confined to the rural district. Rates in excess of 10 per 100,000 were recorded, in 1932, in Chattanooga (12.9), and Memphis (10.6); and in Knoxville and Atlanta the rate was 9.8 and 9.5, respectively. Other cities where the typhoid mortality was well in excess of that for the population of the United States were New Orleans, Nashville, Dallas, Oklahoma City, and El Paso. The figures for Nashville are unique, in that they show that the mortality in the white population was nearly double that among the Negroes; in New Orleans the typhoid fever deathrate among white persons closely approached that for the colored population.—*Bulletin Metropolitan Life Insurance Co.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 20011

PRESENTATION OF CASE

DR. AUGUSTUS S. ROSE:* A seventy year old unemployed chauffeur entered the Outpatient Department complaining of loss of weight and shortness of breath.

Four months before admission the patient noticed slight dyspnea, gradually increasing in severity and most marked on going upstairs. There was no cough, sputum, hemoptysis or wheezing. There had never been any substernal or referred pain. Recently there had been loss of appetite but no vomiting. His bowels had been fairly regular, requiring a light cathartic about every other week. His diet was well balanced. For the past four months he had had nocturia three or four times at night. There was no dysuria or hematuria. He had lost thirty pounds in weight during the past year.

The family history is irrelevant.

He had been married thirty-three years. His wife was living and well. She had had three children and one miscarriage due to accident.

He had typhoid fever thirty-three years before entry, pleurisy forty years before entry and rheumatism twenty-eight years before entry but no swelling of the joints. He had a concussion from a fall on the ice in his youth.

He smoked a pipe continually and used a moderate amount of alcohol daily.

On physical examination the patient was poorly developed and emaciated. The pupils reacted sluggishly to light and accommodation. The fundi showed hazy disks. All the teeth were missing. The left chest was nearly fixed on respiration, though there was some excursion of the diaphragm on percussion; but the right side seemed to compensate. The lungs were negative. The heart was not enlarged; no thrills or murmurs, no increased supracardiac dullness. The blood pressure was 120/80. Liver dullness, fourth space anteriorly on the right. Abdomen: there was a sensation of fullness in the midepigastric to the right epigastric region, and a palpable mass on further examination. There was no tenderness or spasm. The mass moved with respiration but was not freely movable. It measured approximately 10 centimeters in diameter. There was a hard, freely movable non-tender lump on the left scrotum.

A gastro-intestinal x-ray series was negative. A chest film showed calcification in the left axillary pleura, bilateral apical infiltration and fibrosis, a straight left diaphragm and a bulge in the right diaphragm. The right costophrenic angle however was clear. The right diaphragm moved well with respiration. The findings were those of calcification of the pleura, bilateral apical fibrosis, arteriosclerosis, tortuosity of the aorta and fixation of the left diaphragm.

Laboratory examination of the urine was negative except for a very slight trace of albumin and a rare white blood cell.

The patient was seen two weeks later in the Pulmonary Clinic. Nothing further was added to the previous diagnosis of healed tuberculosis. A urological consultant found the prostate not enlarged and very definitely benign. There was no suggestion of cancer.

The patient was admitted to the wards approximately five months after his last visit to the Outpatient Department because of coma for the past five days.

Since his discharge from the Outpatient Department there had been very little change in his condition until three weeks before admission, when he began to make irrational remarks and it was fairly obvious that he was having transient episodes of disorientation. Five days before entry he began to have occasional visual and auditory hallucinations and two days later slurring speech. Since then he had been semistuporous and had taken no food or fluids. He had no headaches, paresthesia or paralysis.

Physical examination was practically the same as in the Outpatient Department. The blood pressure was 90/60. The abdominal fullness was still present, with slight spasm in the right upper quadrant and definite tenderness upon moderate pressure. The biceps reflexes, knee jerks and ankle jerks were present but weak.

The temperature on admission was 99.2°, the pulse 90. The respirations were 26.

The urine showed a trace of albumin, a specific gravity of 1.010, and was otherwise negative. Blood: the white cell count was 55,000, 95 per cent polymorphonuclears, the red cell count 4,170,000. A lumbar puncture showed a very low pressure but good rise on jugular compression. The fluid was clear, colorless and negative. The non-protein nitrogen was 41 milligrams. The carbon dioxide combining power was 54.5 volumes per cent. A fasting blood sugar was 105 milligrams. The blood chlorides were 5.96 milligrams.

A surgical consultant believed that thrombosis was the source of the high white blood count but did not think that the abdomen was the site of disease.

*Senior interne on the East Medical service.

The patient remained in a semistuporous state, looking moribund, and developed a coarse rattle in his throat. His white blood count rose to 67,000 two days after admission. On the third day the abdominal tenderness was generalized, with resistance but no real spasm. He was given daily injections of 2.5 per cent glucose subcutaneously. On the fourth day he became comatose and died.

Differential Diagnosis

Dr. Arlie V. Bock: The total history as given here leaves us with a very confused picture as to the probable cause of death. There are various complicating statements in the record.

We have to take into account in the first place that he was seventy years old and that the presenting symptoms were loss of weight and shortness of breath. These can be due to a great many factors. The factor of marked loss of weight practically rules out his heart as a cause of shortness of breath, and we have to consider as a cause of these presenting symptoms severe anemia, primary anemia or a secondary anemia due to blood loss or some type of leukemia; or, going to the lungs, chronic pulmonary tuberculosis, bronchiectasis or possibly malignant disease in the lung. Then one has to think of the possibility of malignant disease anywhere in the body reaching an end stage, producing anorexia and a generally hopeless physical state.

The remarks to the effect that he had no cough, sputum, hemoptysis or wheezing seem to me to rule out pathology in the lung as a cause of the condition. If he had an old tuberculosis that suddenly flared up we should have more facts than these. The same is true of bronchiectasis. It does not exclude the possibility of a primary carcinoma of the bronchus.

The story that his diet is well balanced does not agree with the statement further down that he had no teeth. Presumably some of his weight loss was due to the fact that his diet was inadequate. I might have mentioned that he was an unemployed chauffeur. If he had been having inadequate food because of unemployment, that may have been a factor in the loss of weight.

Typhoid fever thirty-three years before entry has only the possible significance that he may have developed gall stones subsequently.

His physical examination suggests the possibility of his trouble being in the lung, but there does not seem to be any definite evidence that whatever change he has in his lung is anything of a chronic type which he has had for many years, and I doubt if it plays any rôle in the present picture.

The heart is normal.

The liver dullness on the right is somewhat high.

I think the restriction of the left chest, the change in the size of the left chest and the calcified pleura as shown in this x-ray probably have nothing to do with the present history of shortness of breath.

There seems to be a disagreement as to whether or not a mass was present in the abdomen. One examiner found a palpable mass to the right of the epigastrium and subsequent observers found no evidence of such a mass.

It seems to me the only really significant thing is the x-ray examination. Aside from the old findings is this peculiar bulge in the right diaphragm, which conceivably might be due to a weak diaphragm with a little tendency to herniate through, or to adhesions. If there were adhesions causing it, however, one would expect obliteration of the costophrenic space, and I think the mass felt in the abdomen, in conjunction with the total history, is of great significance in this picture.

From the history just prior to the last admission, when he began to make irrational remarks and to have transitional episodes of disorientation, one gets the impression that two things might be in the background. One is the degree of arteriosclerosis that he had, together with whatever influence dehydration might be playing. I do not know from the notes whether the man was having an adequate intake of fluids or not, but in the presence of dehydration at this age he could have this picture. The symptoms are however also consistent with frontal lobe tumor, which would have to be metastatic presumably, and in view of the x-ray findings in the lung it seems very unlikely that he could have a metastatic lesion in the head. I think metastases do occur in the brain rarely without evidence of pulmonary lesions, do they not?

Dr. Tracy B. Mallory: Yes.

Dr. Bock: That is however a very rare condition. On the whole I should be inclined to attribute the cerebral symptoms to arteriosclerosis, emaciation, dehydration, and the general sick state in which this man found himself.

The rest of the notes are confusing. The surgeon apparently did not think there was any pathology in the abdomen. Previous examiners thought there was.

As to the terminal state of coma, I do not know whether he was in coma or was semistuporous. The notes are confusing about that, but I take it that it was not frank coma, that there was some ability to coöperate. This was probably a terminal manifestation.

The high white cell count with a high count of polymorphonuclears is certainly difficult to explain in a man of this age with no more than he shows, no fever and nothing to suggest a cause for a lesion like peritonitis. There is no reason to suspect a ruptured viscus such as perforated duodenal ulcer or gastric ulcer.

It seems likely that the underlying condition is a malignant process involving the liver, but the mass as described in the right epigastrium

is a little difficult to tie up with the liver. It might easily be a mass in the stomach, but that seems to be ruled out by the gastro-intestinal series. A mass in the pancreas as palpable as this seems to have been at one time would easily give enough compression on the common duct to produce jaundice. The only other lesions that I can think of in the epigastrium are the possibility of aneurysm—and he is too old for that—and a large cyst of the pancreas which might not be giving pressure on the common duct.

I cannot make a diagnosis on the case, but my belief is that he had a malignant process in the liver and that the chances are that the abnormality in the diaphragm was caused by a metastatic portion of it. Whether he had a terminal peritonitis I do not know. We have seen terminal leukocytoses without any adequate explanation, and we have not been able to account for it. The tenderness over the abdomen suggests peritonitis, but if it is peritonitis I am at a loss to explain the origin of it. After taking everything into account I cannot even guess at a diagnosis except for the probable finding of malignant disease involving the liver.

Clinical Discussion

Dr. George W. Holmes: As Dr. Bock pointed out, we have evidence of an old tuberculous process with calcification of the pleura that may have some bearing on this deformity.

Deformities of the diaphragm are due to a number of causes. It is perfectly possible that he has a mass in his liver, either gumma or metastatic malignancy, which is pushing the diaphragm up and giving the appearance seen. A local atelectasis in the lung with a thin flabby diaphragm will produce such a picture. Adhesions to the pleura will do it, and it is not necessary for them to extend into the costophrenic sinus. Adhesions can produce deformity in the outline of the diaphragm. This man has had tuberculosis, and we have to consider adhesions, although this is not the deformity one expects with adhesions. Then we may have difference in the contraction of the muscles of the diaphragm producing double curves and peculiar shapes similar to this. Any deformity of the liver appears when the diaphragm is pulled down hard across it. It is difficult to draw any definite conclusions from deformities of the diaphragm alone. This one is quite marked, and from the story one would agree with Dr. Bock that this ought to be seriously considered in the diagnosis.

The movement of the diaphragm in these cases is of importance. A non-inflammatory mass as a rule will not interfere with respiratory motion. An inflammatory mass causing this deformity would cause definite limitation in the excursion of the diaphragm. The lung markings are unusually large and bright and the diaphragm low, which I think would be against peritonitis. A man with even local peritonitis is apt to have elevation of the diaphragm rather than a low diaphragm.

He has a rather small heart shadow. That may be due in part to the low position of the diaphragm with rotation of the heart. He also has a prominent aortic knob, which is characteristic of arteriosclerosis.

I would agree with Dr. Bock again that if this is a mass in the liver it is probably not gumma but is more likely to be a metastatic nodule or an anatomic variation.

Clinical Diagnoses

Generalized arteriosclerosis.
Mesenteric thrombosis?
Peritonitis?

Anatomic Diagnoses

Primary carcinoma of the intrahepatic bile ducts with metastases to retroperitoneal lymph glands.
Acute typhlitis.
Healed apical tuberculosis, bilateral.
Fibrous and calcareous pleuritis, left.
Hydrothorax, right.
Fibrous pleuritis, right.
Tracheitis and bronchitis, slight.
Ascites, slight.
Arteriosclerosis, moderate coronary, moderate to marked aortic, moderate renal.
Benign prostatic hypertrophy.

Pathologic Discussion

Dr. Mallory: There is no question that the data both on the history and the physical examination are inadequate for a diagnosis in this case beyond the point which Dr. Bock reached.

The patient had malignancy of his liver and it turns out to be a primary carcinoma in the liver, not a hepatoma but a carcinoma of the intrahepatic bile ducts. Nearly all primary cancers of the liver, whether hepatomas or bile duct carcinomas, arise in cirrhotic livers, and this man had a markedly cirrhotic liver due to hemachromatosis, which was quite marked. The pigmentation did not extend however to his skin, so that there was no reason to suspect it clinically, and he did not have sugar in the urine at any time.

Dr. Richard C. Cabot: Was there anything in his brain?

Dr. Mallory: We did not have a chance to examine that.

This is a cross section of the liver. There is the large nodule rising from the superior surface just where the x-ray films showed the irregularity in the diaphragm. That x-ray film was taken several months before his death and

at autopsy the tumor was undoubtedly much larger than it was at the time of this examination.

DR. BOCK: Was no more said in the x-ray interpretation of that film than was put in this record?

DR. MALLORY: No.

DR. HOLMES: Did he have esophageal varices?

DR. MALLORY: None that we were able to find. I think there is no question that we miss slight varices at autopsy. They collapse and are much less obvious then than they would be during life.

This microscopic section of the liver shows a moderate though not very severe cirrhosis with large amounts of yellow-brown pigment in the liver cells and stroma. In this second section it has been stained black with ammonium sulphide.

The tumor itself is a very scirrhous affair, with much connective tissue and relatively few tumor cells. What cells one finds have a tendency to be in glandular arrangement and they bear no resemblance in either their arrangement or appearance to liver cells. It is obviously an adenocarcinoma, and as a very careful search was made for a possible primary focus everywhere in the body, I think we are safe in saying it is a primary bile duct carcinoma on the basis of long standing hemochromatosis.

DR. BOCK: Can you explain his terminal leukocytosis?

DR. MALLORY: Only partially. We found in the cecum an area of bright red injection. The appendix was perfectly normal. The cecum was acutely inflamed. There were no thrombi in the vessels running either to or from this area. There was no mesenteric thrombosis. One has to call it an acute typhilitis, which we see once in a while with a negative appendix.

DR. CABOT: Certainly terminal leukocytoses unexplained at autopsy are common. You do not need an explanation.

DR. MALLORY: Yes, but sixty thousand is pretty high.

DR. BOCK: That needs an explanation, but we do not know what it is.

DR. CABOT: We often do not.

DR. MALLORY: It is not impossible that there was enough necrosis in this tumor to be partially responsible, although one does not usually see counts over twenty thousand with that.

DR. BOCK: Would you agree that the cerebral condition might be a question of malnutrition, dehydration and arteriosclerosis rather than a possibility of a frontal lobe tumor?

DR. MALLORY: I think that is perfectly possible. The patient was practically moribund on the final entry. I do not believe we need much more explanation than that.

CASE 20012

PRESENTATION OF CASE

DR. AUGUSTUS S. ROSE:* *First admission.* A forty-seven year old American Hebrew newspaper man entered for the first time four years and a half before his final admission with the complaint of pain in the chest and left arm of three days' duration.

Four days before admission while at lunch he had a sudden sharp pain in the midsternum and left arm. The pain first appeared in the chest but very quickly became evident in the arm, although there was no radiation. The pain became very severe and "felt like it was trying to break through". This attack was associated with sweating and nausea but no vomiting. He was taken to an emergency relief station where he was put to bed and given a hypodermic. He remained at this hospital for two days and then returned home. On the night before admission shortly after he went to bed the pain suddenly recurred in both the midchest and arm. He was taken to the Emergency Ward of this hospital and during the examination he had three more similar attacks. He was given nitroglycerin, which appeared to lessen the pain a great deal. He was transferred to the wards.

His family history is irrelevant. He had always done hard work. He had been a pugilist for eight years, but gave it up twenty years before entry. He smoked six or eight cigars a day and consumed only moderate amounts of alcohol, never excessive. For the past three years he had worked in the mailing room of a newspaper establishment.

He had gonorrhea at twenty-six which was apparently cured in one year under treatment. Two years later, however, he was operated on at a hospital for stricture. At about the same time he developed arthritis which he was told was due to gonorrhea. He was treated with serum injections and since then had been apparently well.

Physical examination. He was a fairly well developed and nourished man. There was no cyanosis or pallor of the mucous membranes. The heart was not enlarged. The sounds were regular and of fair quality. There were no murmurs. The blood pressure was 140/105. He had an attack during the examination, relieved by nitroglycerin. The blood pressure fell to 115/80. The heart sounds were louder and of better quality. Half an hour later the blood pressure returned to 140/105.

Examination of the urine was negative. Examination of the blood showed a red cell count of 5,500,000, 94 per cent hemoglobin, and a white cell count of 13,500 with 65 per cent poly-

*Senior interne on the East Medical service.

morphonuclears. Examination of the stools was negative. The non-protein nitrogen was 34 mgm. The Hinton and Wassermann tests were negative.

An electrocardiogram showed normal rhythm, rate 110, inverted T_2 and T_3, left axis deviation and slight slurring of the QRS complex in lead II.

The temperature was 99.1°, the pulse 90. The respirations were 20.

He remained in the hospital for nine days and was discharged to his home, which was very near by, to remain in bed for at least another week.

History of interval. After his discharge he was followed in the Cardiac Clinic. He led a limited life and took very good care of himself. He had occasional substernal discomfort with left ulnar paresthesia, too short however to take nitroglycerin. These attacks increased in frequency. It was felt in the Cardiac Clinic that his heart was getting larger. His blood pressure gradually rose to 175/120 and he developed some dyspnea on exertion. Two weeks before readmission he had a more severe attack which was relieved by nitroglycerin. On the day of readmission, while at work, he had the most severe attack since the previous year. This attack was not relieved by two tablets of nitroglycerin.

Second admission, fifteen months after his previous discharge.

Physical examination showed that he was in some distress and was anxious. His lips were cyanotic. The heart was slightly enlarged; the measurements were midclavicular line 9.5 centimeters, supracardiac dullness 6.5, right border of dullness 4, left border 10. The blood pressure was 176/120. There were no murmurs. There were râles at both bases. There was no peripheral edema.

The temperature was 98°, the pulse 93. The respirations were 21.

Two days after admission his temperature rose to 100°. His white blood cell count was 18,900. There was a mild fall in blood pressure. The heart sounds were feeble. No friction rub was heard. He was discharged in twenty days.

History of interval. For the first month following discharge he got up very gradually and in the second month he started back to work. He then began to have a few attacks which were always relieved by nitroglycerin. Most of the pain was tingling or limited to the left forearm. On the day before admission he had an attack during an argument at a ball game which was relieved half an hour later after he had gone home in order to get nitroglycerin. The pain recurred however and he entered the Emergency Ward.

Third admission, three months after his second discharge.

Examination was essentially the same as on the previous admission. The blood pressure was 185/120.

Digitalization was maintained. When first seen he was fibrillating. This lasted for twelve hours, then gallop rhythm set in and lasted for two or three days. The heart then became slow and regular.

His white blood cell count, which on admission was 13,000, dropped to 7,000. An electrocardiogram showed normal rhythm, rate 90, left axis deviation, diphasic T_2 and inverted T_3.

He was discharged much improved at the end of three and a half weeks.

History of interval. After discharge his condition remained about the same. Even moderate exertion produced anginal attacks, relieved by nitroglycerin. He had no other cardiac symptoms however. He took digitalis a grain and a half daily. For a week before readmission his pain was more severe than usual and not so readily or completely relieved by nitroglycerin. On the morning of admission at breakfast he developed a very severe pain which radiated down his left arm to the first two fingers. Associated with this was choking substernal distress which radiated to his right shoulder. This was relieved only by morphia after three hours.

Fourth admission three years after his third discharge.

Examination showed slight cyanosis of the lips. There were fine and medium moist râles in the lower halves of both lungs posteriorly and slight dullness at the left base. The heart was definitely enlarged to the left, being 12 centimeters to the left of the midclavicular line. The blood pressure was 160/120. The sounds were of only fair quality, regular, no gallop rhythm. There was slight precordial tenderness.

The white blood cell count was 10,350, 77 per cent polymorphonuclears.

An electrocardiogram showed normal rhythm, rate 80, inverted T_1, diphasic T_2, slurred and widened QRS and left axis deviation.

He remained in the house for three weeks and was discharged greatly improved, with a blood pressure of 115/80.

History of interval. After discharge he was kept on digitalis three grains daily, nitroglycerin and luminal. Although he was on limited activity he began to have attacks of choking and of coughing and anginal attacks at night, forcing him to sit up most of the night. He had occasional attacks of nocturnal dyspnea, which became more severe two months before admission. Five weeks before admission he developed swelling of the ankles for the first time. Three weeks before admission he began to have upper abdominal and substernal non-radiating pain which was worse upon exercise and after meals. These

attacks were not at all like his old anginal attacks. They were relieved by soda. The edema gradually became more marked and for two weeks before his fifth admission he noticed some swelling of the abdomen.

Fifth admission, four months and a half after his fourth discharge.

Physical examination showed a slightly wasted middle-aged man in no distress. There was slight cyanosis of the lips. The radial vessels were slightly thickened. The blood pressure was 160/116. There was no change in the heart findings. The abdomen was moderately distended. The liver was felt four to five fingerbreadths below the costal margin and was slightly tender. There was dullness in the flanks with slight fluid wave. There was moderate edema of the lower extremities and sacrum.

The urine examination showed a slight trace of albumin and six to eight white blood cells. The red blood cell count was 6,300,000, the hemoglobin 80 per cent. The white cell count was 10,400, with 74 per cent polymorphonuclears. A phenolsulphonephthalein test showed 45 per cent excretion in one hour.

During the first week in the hospital he was given three injections of salyrgan, which failed to relieve the edema. On the ninth day his temperature rose to 103°, the pulse to 115. Three days later the temperature reached 107°, the pulse 128 and the respirations 52. The nonprotein nitrogen was 60 milligrams. He rapidly failed after this and died on the thirteenth day.

Dr. George W. Holmes: The film that we have was taken at the first admission and was interpreted at that time as not showing anything definitely abnormal. I would agree with that interpretation except to add that possibly the aortic knob is a little prominent for a man of that age.

Differential Diagnosis

Dr. William B. Breed: There are two comments I should like to make about the past history in this case. One of them is about the statement that he had always done hard work and had been a pugilist for eight years prior to twenty years before admission, which would indicate that he led a rather strenuous physical life. Also, the person who made the excerpts thought we would be interested in the venereal aspect and mentioned that he had had severe gonorrhea and had been treated with "serum injections" at one time. Perhaps that was to attract my attention towards the question of syphilis, but I think that there is very little evidence on physical examination, in the history, or apparently in the x-ray films, which I had not seen before, that he had luetic aortitis or an aneurysm.

I think it is fairly obvious that he had angina pectoris; but of course angina pectoris literally translated means nothing more or less than pain in the chest. We have interpreted it as a characteristic precordial pain with radiation to the right or the left arm. The onset of this man's first attack certainly does not sound like the clinical description of angina pectoris as such. It is very much more suggestive of a coronary occlusion which does produce pain in the chest, indicating a faulty functioning of the coronary vessels. I think he undoubtedly had at least three or four occlusions with cardiac infarct and between such occlusions rather persistently the symptom complex of angina pectoris on the basis of his coronary disease.

His first electrocardiogram apparently was taken before there was any digitalis given and that certainly indicates that there is some abnormality in the coronary circulation, and the last electrocardiogram shows more than could be accounted for on the basis of digitalis poisoning. There is a slurred and widened QRS along with left axis deviation and diphasic T_2.

As to the question of fibrillation, Dr. Rose said that he was suspected of having it. The association of auricular fibrillation with angina pectoris, especially in a case of short duration, is not common. It does occur, but I should be more inclined to think that the irregularity was not true auricular fibrillation at that time.

What did he die of? At the end he had a very high temperature. According to the record I should be inclined to think that he did not die of a terminal large coronary occlusion but rather from pulmonary infarcts, pneumonia, or massive cerebral hemorrhage. You note that his temperature rose to 107°, his pulse to 128 and the respirations to 52. I should think his was probably not, as I said, primarily a coronary or a cardiac infarct death. I should expect to find that he had a large arteriosclerotic heart with probably a number of infarcts, old infarcts and perhaps one more recent one occurring at the time of his last admission; and a good deal of pathology in the lungs, either pneumonia alone or pulmonary infarcts.

Dr. William D. Smith: I saw this man at two of his admissions. I did not see him at the time he died. There is not very much to add. Dr. Breed has covered the case pretty well. I think that the first time I saw him the patient had hypertensive and arteriosclerotic disease, that his symptoms were due to coronary disease quite definitely, and that he probably had had two or three small coronary infarcts in the past. The last time he came in I felt that he had another coronary accident and congestive failure.

Whether or not he had pathology in the lungs at the end I do not know. I should think it unlikely that he had a cerebral hemorrhage because his temperature went up rather gradually and there is nothing in the record to suggest a cerebral accident. He might well have had terminal pneumonia or pulmonary infarcts.

DR. TRACY B. MALLORY: Have you anything to say, Dr. Richardson?

DR. WYMAN RICHARDSON: I have very little to add. I saw him simply in the terminal stages of the disease. I was interested in his high temperature, which I attributed purely to his cardiac failure. I think it is an interesting question as to the cause of the temperature in cardiac failure. I believe it is supposed to be at least partly due to the failure of the heat regulating mechanism due perhaps to failure of the peripheral circulation.

DR. HOWARD B. SPRAGUE: I think I followed this man more closely in the last five years than anyone else. There are two or three points to bring out. One is that he was an extremely nervous man whose symptomatology was difficult to assess. He had from time to time definite findings indicating coronary occlusion that made us believe he had progressive coronary disease in spite of the confusing elements of the neurosis.

Another point is that he did not have, as I remember it, at any time very characteristic electrocardiograms indicating massive infarct.

One other point is that I felt he was a patient who had a rare type of anginal equivalent, that is regurgitation of food, or vomiting after eating, which is comparable to the belching that one sees in cases with coronary disease.

CLINICAL DIAGNOSES

Arteriosclerotic heart disease with congestive failure.
Angina pectoris.

ANATOMIC DIAGNOSES

Coronary sclerosis with multiple areas of occlusion.
Cardiac infarction, old and recent.
Pulmonary infarcts.
Acute bronchitis.
Chronic vascular nephritis.

PATHOLOGIC DISCUSSION

DR. MALLORY: The autopsy on this man showed a large area of infarction not in the usual place near the apex of the left ventricle, but on the posterior wall of the left ventricular wall up towards the base; and corresponding with that the point of maximum narrowing in the coronary branches was not in the descending branch of the left coronary but in the circumflex branch of the left with a second one in the corresponding branch of the right, which came over very close to the edge of the infarct. Both these branches were extremely narrow. There was a point where there was hardly any visible lumen left. There was however fluid blood in each vessel that could be expressed with slight pressure, and there was no evidence of any fresh thrombosis. In other words, he had extreme narrowing of one branch of the left and one branch of the right coronary, but no complete occlusion at any point in either artery.

The area of infarction showed two distinct zones, a central one which was fibrous and evidently old and a peripheral one which showed hemorrhage and was obviously quite fresh, I think coincident with his last entry to the hospital. I believe it is fairly common for multiple infarctions to appear at approximately the same area in the heart, so that even when a clinical history suggests two or three infarcts we may be able to find only one, since the various lesions have been superimposed and the scars more or less merged.

The lungs showed several infarcts, relatively small ones. The largest was about 5 centimeters in diameter. There was no pneumonia.

We were not allowed to examine the head, so I do not know whether there was any cerebral accident.

A PHYSICIAN: Will you say more about the superimposition of one infarction upon another? What happens? What is the mechanism?

DR. MALLORY: There are several reasonable hypotheses to cover such a situation. In the first place infarction does not necessarily destroy all the muscle cells in an involved area, in fact the distribution is often peculiarly irregular. Hence a second infarction might easily involve the remaining muscle cells of exactly the same area. Probably however necrosis of another area in immediate apposition with the first is more usual. Several vascular mechanisms might be imagined which would cover such situations. A completely thrombosed vessel not infrequently recanalizes to a significant extent. A new thrombosis of the canalizing vessel would result in a second infarct of closely similar distribution. Moreover the coronary arteries are not anatomically speaking end arteries, hence collateral circulation always limits and occasionally prevents infarction in the face of thrombosis. Sufficient narrowing of the collateral branches might extend the area of necrosis. Finally it is by no means impossible that infarction may occur without thrombosis, simply from prolonged spasm. If this is true, another spastic episode or a final thrombosis would again lead to superimposed or nearly superimposed infarcts.

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OF WHAT SHALL DIABETICS DIE?

MASSACHUSETTS holds an enviable record in its reduction of the mortality of diabetic coma. Whereas formerly, both here and abroad, 60 per cent of all diabetic deaths were due to coma, the rate in Boston for the first 11 months of 1933 was 11 per cent. The rate in a city considerably larger than Boston was, according to a recent compilation, more than three times as great. So too, the Boston proportion of diabetic deaths due to gangrene was 15 per cent in contrast to 23 per cent in the other city.

So far so good, but we should not be content. No diabetic should die from coma and much fewer than at present should die from gangrene, because that, too, is largely preventable. The further and more energetic application of the same methods, which have lowered the mortality of these diabetic complications, will accomplish much more. Education in diabetic fundamentals is the outstanding need, and it is only by teaching line upon line, precept upon precept, that success will be achieved.

But of what can our diabetics die? They are not immortal. Necessarily more will die of cancer, although that is not an enviable end result of a faithful diabetic life. Perhaps it should be less common with diabetics than with non-diabetics, because in diabetics under close observation earlier diagnoses should be made and in consequence more cures should be expected. Unfortunately more are dying of tuberculosis and the attention of our readers is called to the series of articles beginning in this number on this new diabetic menace which was the old diabetic menace of the last century. It appears that now tuberculosis in diabetics is two to three times as frequent as in non-diabetics, that it is ten times as common in diabetic children, that eight per cent of patients rescued from coma within three years develop it and most unfortunately that tuberculosis in a diabetic is almost never recognized in an early stage. More x-ray films should be taken of diabetic chests, for when the diagnosis of tuberculosis is made reasonably early, the modern diabetic often shows surprising improvement under treatment.

What then is left to the diabetic? Pneumonia, the old man's friend, and arterial changes chiefly in the heart and brain which often are sudden and not seldom painless. Indeed, the latter are the complications to which the oldest inhabitants in our midst usually succumb. The diabetic's aim should be to avoid their premature development and so attain a ripe old age. Actually this is being accomplished because now the average diabetic age at death is about 63 years instead of 44 years, the average age at death of diabetics prior to 1915.

PAGE THE SPECIALIST IN FRACTURES!

ON December 1, 1933 there was reported in the *Waltham News-Tribune,* an accident of which the scene was not mentioned. Doubtless it will be brought to the attention of the proper authorities for investigation, but it is easily ascertained that a fracture of a sort had been sustained. Exceeding the speed limit was the cause.

The notice in the newspaper reads as follows: "*Middlesex College Notes*—The results of the May examination given by the Massachusetts Board of Registration in Medicine published this week in the *American Medical Journal* showed that more graduates of the Middlesex College of Medicine and Surgery than of any other medical college in the country were successful in passing the examination for licensure in this state."

Careful search for the *American Medical Journal* fails to disclose a copy, but in the *Journal of the American Medical Association* for November 25, 1933, under the heading "Massachusetts May Examination" are published some figures which are probably what the reporter for

the *News-Tribune* had in mind. These figures reveal the truth before the accident, and there was found a similar portrait, full length, in the archives, in an obscure and little known publication, marked Public Document 56, otherwise known as the Annual Report of the Board of Registration in Medicine.

In the *Journal* there appears also a list of candidates, registered by "endorsement". It is a misleading term because each such candidate has the following notation, "Endorsement of the National Board of Medical *Examiners.*"

Turning to the Massachusetts statute one finds that "In determining the qualifications necessary for registration as a qualified physician, the board may at its discretion accept the certificate of the National Board of Medical Examiners of the United States chartered under the laws of the District of Columbia in place of and as equivalent to its own professional examination." Such candidates are therefore licensed by "passing the examination for licensure in this state", and the statement in the *News-Tribune* is not quite true.

The "whole truth", unfractured, as noted in the *Journal*, so far as it can be put down in figures is that of twenty-five graduates of Middlesex who took the examination given by the Massachusetts Board in May 1933, nineteen were rejected and six were registered; of seven Tufts graduates who took this examination, two were rejected and five were registered; of five Harvard graduates who took this examination not one was rejected and five were registered; and several National Board diplomates also were registered. If one does not reckon in these diplomates, about four-fifths of the Middlesex graduates were rejected, one-third of the Tufts graduates and none of the Harvard graduates. In May 1933, more graduates from Middlesex College of Medicine and Surgery were rejected by the Massachusetts Board of Registration in Medicine than from any other of the twenty-eight medical schools whose graduates came before the Board, even more than all the osteopaths who failed to become registered.

But there are the National Board diplomates. The figures for the period covered by the May examination, the dates of which do not coincide with the dates given in the *Journal*, have been secured through the courtesy of the Board of Registration in Medicine and are as follows: Harvard 11, Tufts 4, Middlesex 0. The graduates of Middlesex are not eligible to take the examination given by the National Board. Therefore on this basis the figures are Harvard 16, Tufts 9, Middlesex 6.

In the month of May 1933, of diplomates of the National Board, there were registered five graduates of Harvard, one graduate of Tufts and again none from Middlesex. The score then is Harvard 10, Tufts 6, Middlesex 6.

This study does not indicate the scene of the accident, whether in Waltham or in some other place, but it shows precisely the seat of the fracture.

It is to be regretted that there should be circulated in the press misleading and inaccurate reports about a medical school which has been chartered by the Commonwealth of Massachusetts even though the graduates of this school are not eligible to take the examination of the National Board, and are reported to be ineligible in every state outside of Massachusetts to take examination for license to practice medicine.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ROOT, HOWARD F. A.B., M.D. Harvard University Medical School 1919. Physician, New England Deaconess Hospital. Assistant in Medicine, Courses for Graduates, Harvard Medical School. His subject is "The Association of Diabetes and Tuberculosis. Epidemiology, Pathology, Treatment, and Prognosis." Page 1. Address: 81 Bay State Road, Boston.

NISSEN, H. ARCHIBALD. A.B., M.D. Harvard University Medical School 1916. Assistant Physician, Robert B. Brigham Hospital. Member of the Staff and Visiting Physician, New England Deaconess Hospital. Assistant Physician, Palmer Memorial Hospital. Former Instructor in Medicine, Harvard Medical School. Address: 205 Beacon Street, Boston. Associated with him is

SPENCER, K. A. Survey Executive, Robert B. Brigham Hospital, Boston. Address: 61 Wait Street, Boston. Their subject is "Sugar Tolerance in the Arthritic." Page 13.

WHITE, PAUL D. A.B., M.D. Harvard University Medical School 1911. Physician, Massachusetts General Hospital. Assistant Professor of Medicine, Harvard Medical School. His subject is "Cardiovascular Review for 1932." Page 20. Address: Massachusetts General Hospital, Boston.

The Massachusetts Medical Society

POSTGRADUATE INSTRUCTION

SCHEDULE FOR DISTRICT MEDICAL SOCIETIES

The Executive Committee has arranged the following schedule for the completion of the postgraduate extension courses for 1933 and 1934:

Barnstable

(Hyannis on Sundays at 4:00 P.M.)

Date Course

January 14, 1934—No. 5—Acute Infections.
January 21, 1934 — No. 8 — Medical and Surgical Emergencies.
January 28, 1934—No. 2—Vascular Disease.
February 4, 1934—No. 1—Practical Medical Principles for Everyday Use.
February 11, 1934—No. 3—Gastro-Intestinal Disease (A).
February 18, 1934—No. 15—Endocrinology.
February 25, 1934—No. 18—Vaccine Therapy.
March 4, 1934 — No. 4—Gastro-Intestinal Disease (B).
March 11, 1934—No. 19—Arthritis.
March 18, 1934—No. 11—Problems of Infancy and Childhood.

Bristol North

(Attleboro—Sturdy Hospital on 2nd and 4th Tuesdays of January and 1st and 3rd Tuesdays of other months, at 3:00 P.M. Nurses' Lecture Room.)

January 9, 1934—No. 1—Practical Medical Principles for Everyday Use.
January 23, 1934—No. 2—Vascular Disease.
February 6, 1934—No. 3—Gastro-Intestinal Disease (A).
February 20, 1934—No. 4—Gastro-Intestinal Disease (B).
March 6, 1934—No. 5—Acute Infections.
March 20, 1934 — No. 8 — Medical and Surgical Emergencies.
April 3, 1934—No. 10—Pathological Pregnancy.
April 17, 1934—No. 14—Syphilis and Dermatology.
May 1, 1934—No. 15—Endocrinology.
May 15, 1934—No. 19—Arthritis.

Bristol North

(Taunton—Morton Hospital on Thursday of each week at 7:30 P.M.)

January 11, 1934—No. 1—Practical Medical Principles for Everyday Use.
January 18, 1934—No. 2—Vascular Disease.
January 25, 1934—No. 3—Gastro-Intestinal Disease (A).
February 1, 1934—No. 4—Gastro-Intestinal Disease (B).
February 8, 1934—No. 5—Acute Infections.
February 15, 1934 — No. 8 — Medical and Surgical Emergencies.
March 1, 1934—No. 10—Pathological Pregnancy.
March 8, 1934—No. 11 — Problems of Infancy and Childhood.
March 15, 1934—No. 15—Endocrinology.
March 22, 1934—No. 19—Arthritis.

Bristol South

(Fall River—Truesdale Hospital on Wednesday of each week at 4:00 P.M.)

January 10, 1934—No. 1—Practical Medical Principles for Everyday Use.
January 17, 1934—No. 2—Vascular Disease.
January 24, 1934 — No. 3—Gastro-Intestinal Disease (A).
January 31, 1934 — No. 4—Gastro-Intestinal Disease (B).
February 7, 1934—No. 5—Acute Infections.
February 14, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
February 21, 1934—No. 15—Endocrinology.
February 28, 1934—No. 18—Vaccine Therapy.
March 7, 1934—No. 19—Arthritis.
March 14, 1934—No. 22—Diabetes and Vitamins.

Bristol South

(New Bedford—St. Luke's Hospital on Friday of each week at 4:00 P.M.)

January 12, 1934—No. 1—Practical Medical Principles for Everyday Use.
January 19, 1934—No. 2—Vascular Disease.
January 26, 1934—No. 3—Gastro-Intestinal Disease (A).
February 2, 1934—No. 11—Problems of Infancy and Childhood.
February 9, 1934—No. 5—Acute Infections.
February 16, 1934 — No. 8 — Medical and Surgical Emergencfes.
February 23, 1934—No. 10—Pathological Pregnancy.
March 2, 1934 — No. 4—Gastro-Intestinal Disease (B).
March 9, 1934—No. 15—Endocrinology.
March 16, 1934—No. 19—Arthritis.

Essex South

(Salem on Tuesday of each week at 4:00 P.M. Salem Hospital. Auditorium of the Nurses' Home.)

January 9, 1934—No. 4—Gastro-Intestinal Disease (B).
January 16, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
January 23, 1934 — No. 8 — Medical and Surgical Emergencies.
January 30, 1934—No. 15—Endocrinology.
February 6, 1934—No. 19—Arthritis.

Franklin

(Greenfield—Franklin County Hospital on Wednesday of each week at 8:00 P.M.)

January 3, 1934—No. 18—Vaccine Therapy.
January 10, 1934 — No. 8 — Medical and Surgical Emergencies.
January 17, 1934—No. 4—Gastro-Intestinal Disease (B).
January 24, 1934—No. 3—Gastro-Intestinal Disease (A).

Hampden

(Springfield—Thursday of each week at 4:00 P.M.)
(Holyoke—Thursday of each week at 8:00 P.M.)

January 11, 1934—No. 1—Practical Medical Principles for Everyday Use.

January 18, 1934—No. 19—Arthritis.
January 25, 1934—No. 18—Vaccine Therapy.
February 1, 1934—No. 2—Vascular Disease.
February 8, 1934 — No. 8 — Medical and Surgical Emergencies.
February 15, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
March 1, 1934—No. 4—Gastro-Intestinal Disease (B).
March 8, 1934—No. 3—Gastro-Intestinal Disease (A).
March 15, 1934—No. 15—Endocrinology.
March 22, 1934—No. 5—Acute Infections.

Hampshire

(Northampton—Wednesday of each week at 4:15 P.M.)
January 10, 1934—No. 19—Arthritis.
January 17, 1934—No. 18—Vaccine Therapy.
January 24, 1934—No. 15—Endocrinology.
January 31, 1934—No. 10—Pathological Pregnancy.
February 7, 1934 — No. 8 — Medical and Surgical Emergencies.
February 14, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
February 21, 1934—No. 5—Acute Infections.
February 28, 1934—No. 3—Gastro-Intestinal Disease (A).
March 7, 1934—No. 2—Vascular Disease.
March 14, 1934—No. 1—Practical Medical Principles for Everyday Use.

Middlesex East

(Melrose—Melrose Hospital on Wednesday of each week at 4:00 P.M.)
January 10, 1934—No. 4—Gastro-Intestinal Disease (B).
January 17, 1934—No. 11—Problems of Infancy and Childhood.
January 24, 1934—No. 15—Endocrinology.
January 31, 1934—No. 19—Arthritis.

Middlesex North

(Lowell—St. John's Hospital on Friday of each week at 7:00 P.M.)
January 5, 1934—No. 10—Pathological Pregnancy.
January 12, 1934—No. 19—Arthritis.

Middlesex South

(Cambridge — Cambridge Hospital on Monday of each week at 4:30 P.M.)
January 15, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
January 22, 1934—No. 8—Medical and Surgical Emergencies.
January 29, 1934—No. 11—Problems of Infancy and Childhood.
February 5, 1934—No. 19—Arthritis.

Norfolk

(Faulkner Hospital on Monday of each week at 3:30 P.M.)
January 8, 1934 — No. 8 — Medical and Surgical Emergencies.
January 15, 1934—No. 5—Acute Infections.
January 22, 1934—No. 2—Vascular Disease.
January 29, 1934—No. 19—Arthritis.
February 5, 1934—No. 3—Gastro-Intestinal Disease (A).
February 12, 1934—No. 4—Gastro-Intestinal Disease (B).

Norfolk

(Boston City Hospital—Staff Room on Wednesday of each week at 4:00 P.M.)
January 10, 1934—No. 4—Gastro-Intestinal Disease (B).
January 17, 1934—No. 14—Syphilis and Dermatology.
January 24, 1934—No. 11—Problems of Infancy and Childhood.
January 31, 1934—No. 5—Acute Infections.
February 7, 1934 — No. 8 — Medical and Surgical Emergencies.
February 14, 1934—No. 19—Arthritis.

Norfolk

(Norwood — Norwood Hospital on Friday of each week at 8:30 P.M.)
January 12, 1934—No. 11—Problems of Infancy and Childhood.
January 19, 1934—No. 6—Diseases of the Blood and Hematopoietic System.
January 26, 1934 — No. 8 — Medical and Surgical Emergencies.
February 2, 1934—No. 4—Gastro-Intestinal Disease (B).
February 9, 1934—No. 14—Syphilis and Dermatology.
February 16, 1934—No. 19—Arthritis.

Norfolk South

(Quincy—Quincy City Hospital on Monday of each week at 8:30 P.M.)
January 8, 1934—No. 19—Arthritis.
January 15, 1934—No. 15—Endocrinology.
January 22, 1934—No. 10—Pathological Pregnancy.
January 29, 1934 — No. 8 — Medical and Surgical Emergencies.
February 5, 1934—No. 3—Gastro-Intestinal Disease (A).
February 12, 1934—No. 2—Vascular Disease.
February 19, 1934—No. 1—Practical Medical Principles for Everyday Use.
February 26, 1934—No. 4—Gastro-Intestinal Disease (B).
March 5, 1934—No. 18—Vaccine Therapy.
March 12, 1934—No. 5—Acute Infections.

Worcester

(Worcester—on Wednesday of each week at 7:30 P.M.)
January 17, 1934—No. 3—Gastro-Intestinal Disease (A). (Hahnemann Hospital.)

January 24, 1934—No. 18—Vaccine Therapy.
January 31, 1934—No. 22—Diabetes and Vitamins.

Worcester North

(Ayer—Ayer Hospital on Thursday of each week at 8:00 P.M.)

January 11, 1934—No. 7—Practical Psychiatric Problems.
January 18, 1934—No. 10—Pathological Pregnancy.
January 25, 1934—No. 15—Endocrinology.
February 1, 1934—No. 2—Vascular Disease.
February 8, 1934—No. 19—Arthritis.
February 15, 1934—No. 20—Genito-Urinary Disease.
March 1, 1934—No. 8—Medical and Surgical Emergencies.
March 8, 1934—No. 5—Acute Infections.
March 15, 1934—No. 11—Problems of Infancy and Childhood.
March 22, 1934—No. 1—Practical Medical Principles for Everyday Use.

MISCELLANY

AN ARTIFICIAL ESOPHAGUS

Those who were privileged to hear and see Professor G. Grey Turner of Newcastle upon Tyne when he lectured before the Massachusetts Medical Society in 1931 will be especially pleased to know that this eminent surgeon has succeeded in providing a passage from the upper portion of the esophagus to the stomach after removing a considerable length of the esophagus for cancer of that organ.

This artificial passage, according to the report, was made by utilizing the skin of the chest to form a tube which through an opening in the neck was joined to the esophagus above and, by utilizing a loop of intestine, to the stomach below through which food could be swallowed.

The series of plastic operations needed to establish the communication covered several months and the patient enjoyed the food that was eaten and passed through this detour. This successful series of operations shows that human ingenuity and a coöperative patient indicate that further triumphs of surgery are possible to well-trained and devoted scientists in this field.

RESOLUTIONS RESPECTING AN ARTICLE BY DR. MORRIS FISHBEIN

At a meeting of the Council of the Chicago Medical Society, November 14, 1933, the following resolution presented by Dr. L. E. Day was adopted:

Whereas, Dr. Morris Fishbein, Editor of the *American Medical Association Journal*, has recently (September, 1933) contributed an article on The Costs of High Obstetrical Care, to the *American Mercury*, presumably for hire, and

Whereas, in said article Dr. Fishbein follows the lead of the irresponsible de Kruif in attacking the Medical Profession for personal profit, and

Whereas, said article makes statements incapable of proof and indefensible as subjects for discussion in the lay press, and

Whereas, such derogatory attacks upon the Medical Profession raises (sic) a question as to his fitness for employment by the American Medical Association;

Therefore Be It Resolved, that the Council of the Chicago Medical Society deplores the total lack of propriety exhibited in said article and resents Dr. Fishbein's disloyalty to the profession which employs him as one of its trusted representatives, and

Be It Further Resolved, that this breach of faith be called to the attention of the Board of Trustees of the American Medical Association by sending a copy of these resolutions to the members thereof with the request that proper disciplinary measures be instituted which will make such violations of decency, ethics, morality and common sense impossible in the future.—*Bulletin of the Chicago Medical Society.*

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

Communicable Diseases Among School Children*

BY JAMES A. KEENAN, M.D.

It is desirable that the public have some knowledge of the principles of school hygiene in so far as it relates to the control of communicable disease. With this subject in view, we will endeavor to set before you a few relative facts as well as procedures known and generally carried out in school systems throughout the Commonwealth of Massachusetts.

Closing of schools in the presence of epidemics of communicable diseases was at one time the only method of protection and control. At the present time, this system is resorted to only at a time of an outbreak of a little understood disease.

Nearly all of the diseases that affect the school child are now fairly well understood, first, as to the spread of the disease, and secondly, as to the methods to be followed as a means of preventing the spread of infection. In many cases the cause of contagious diseases affecting children is known. For instance, medical science has proved that the typhoid bacillus causes typhoid fever, that the Klebs Loeffler bacillus causes diphtheria, and that a certain strain of the germ known as streptococcus causes scarlet fever, epidemic adenitis, as well as septic sore throat. Other forms of streptococci cause rheumatic fever, acute appendicitis, virulent pneumonia, acute seriously disabling endocarditis, or valvular heart disease.

The pneumococcus, of which there are at least four identified types, cousins, so to speak, but all

*Station WBZ, November 10, 1933.

reacting in a slightly different manner when tested, is the cause of pneumonia. The meningococcus, an organism that looks somewhat like a biscuit under the microscope and is very difficult to grow artificially, is the cause of meningitis. If a case of epidemic meningitis occurred in a classroom, all the children in that classroom would be examined for the presence of the meningitis germ which may be found in the back of the nose. This examination is performed by wiping some of the secretions of the back part of the nose with a wire wrapped in sterile cotton and the secretions are planted on a suitable food media where they develop at body temperature and can then be detected. Children showing the presence of meningococcus are excluded from school and isolated to protect other pupils and the public, who may be in such a condition of health as to allow the germ to establish itself and cause the disease.

Scarlet fever can be controlled and can be prevented best by school health authorities. In fact, the schools are largely responsible for the decrease in the virulent type of scarlet fever cases. The disease is now so mild that it is often very difficult for an expert to pronounce some cases as scarlet fever. If there is any doubt as to whether a child has scarlet fever, the mistake, if any, is made on the side of the disease. This is to protect the child from the complications resulting from scarlet fever which are many and varied. Once a case of scarlet fever is discovered, the school physician notifies the Health Department which department is invested with very wide police power and sees to it that the child is isolated for at least twenty-eight days. If there are any discharges from the nose or ears, or from open sores, or if there are enlarged glands, the case is not released by the Health Department until such time as the child is well. Desquamation is not of itself capable of spreading scarlet fever. If the desquamating or peeling skin is contaminated with discharges, it is then dangerous. If a child has had scarlet fever, discharges from the nose or ears may be a source of infection long after a period of six weeks has elapsed. Children, even though they appear well, may spread the disease through such discharges. When a child who has been absent after an attack of scarlet fever returns to school, he is examined by the school physician before he is allowed to enter the classroom. If any discharges from the nose, ears, or open sores are present, he is excluded until such time as these conditions disappear. The school authorities of Boston exclude from school all children of the immediate family when a case of scarlet fever occurs in a household for a period of at least seven days after exposure. This is to prevent the spreading of the disease in other classrooms. Children who have had scarlet fever or who react negatively to the Dick test and who are later exposed to the disease are not excluded from school. A well child from a household where a case of scarlet fever is quarantined by the Health Department and who has not had the disease is not allowed to attend school for the full period of quarantine, that is, for at least twenty-eight days. If the case is isolated in a hospital, or if the children of the household who are not immune are transferred to the household of a relative or friend, the exposed children are allowed to attend school after a period of seven days has elapsed from the time of such exposure. This rule alone has, in some schools, cut the number of cases of scarlet fever from fifty to seventy-five per cent. Without school medical inspection and supervision, scarlet fever would be uncontrolled.

Scarlet fever always increases with the advent of the cold weather whether the schools are open or not. Without school medical inspection, nobody would know how much scarlet fever was prevalent, many cases would never be quarantined, there would be a serious spread of the disease, and many more children would be allowed to suffer from a very serious disease, the after-effects of which would in all probability cause grave injury. Everybody knows that scarlet fever cases require three weeks in bed in order to avoid serious complications. Therefore, the discovery of a case of missed scarlet fever in a school child is a very valuable aid in preserving and conserving the child's health.

Measles is a disease occurring commonly among school children. It is one of the most contagious diseases known to man. No person is immune to the disease, young or old; all races are affected in all climates. Measles is a problem in the schools only among the younger children in the kindergartens and first grades. It is a very serious disease unless good medical attention and excellent nursing aid are enlisted. It is possible to detect measles in its early stages. Inside the mouth on the cheek surface appear tiny white spots with a red base. They look somewhat like grains of salt. These spots are often present before the child shows any suggestive signs of measles. When a case of measles is known to have occurred in a classroom, the school physician examines the other exposed children at the end of eight days to detect the presence or absence of these spots. These inspections are carried on for three days. If these spots indicative of measles are found, the children so affected are recommended for exclusion by the school physician and a note is sent to the parent stating that the child presents symptoms suggestive of measles. This method forewarns the mother, allows her to procure adequate medical care and nursing, and minimizes the attack, thus conserving the child's health. If these spots are found early enough, exclusion prevents the other school children from contracting the disease. The reason that inspections of pupils in close contact with measles cases are made by school physicians eight days after exposure to the disease is because it takes from ten to fourteen days from the date the child has been exposed to measles to the date

the disease manifests itself. It can be seen that a scientific knowledge of measles applied can prevent the spread of the disease as well as most happily preventing serious after-effects and modifying the character of the disease when it does occur to a milder form.

If an outbreak of smallpox should occur, the public schools would be the only place where an epidemiologist (a person concerned with the discovery and prevention of disease) could obtain extremely valuable information. Every school child's vaccination history is kept on file and the date of vaccination is always available on the school record card.

Mumps is a disease that may manifest itself twenty-one days after a child has had close contact with a person suffering from this illness. This is the reason that all children from a household where a case of mumps occurs are excluded for twenty-one days. If allowed to return to school earlier, they may develop the disease and thus expose many other children to mumps.

Chicken pox is an acute infectious disease, extremely contagious, commonly occurring in children of school age. The disease usually does very little damage to the human system and is important only in that it may be confused with mild smallpox which disease it resembles very closely. The trained school physician can readily differentiate the diseases smallpox and chicken pox thus enabling us to minimize the incidence of chicken pox, and through the practice of vaccination, prevent smallpox from occurring.

German measles occurs occasionally in epidemic form among school children. The disease is relatively unimportant, usually running a mild uncomplicated course. It is frequently confused with scarlet fever as the rash of a mild case of scarlet fever closely resembles the rash of German measles.

Diphtheria is a disease that usually establishes itself in the throat or in the nose. Due to the campaign carried on in the Boston public schools against diphtheria, principally by the injections with toxin antitoxin, this disease has been almost banished from our school systems.

Schools are, therefore, kept open in times of epidemic because medical science can be applied systematically and universally to the end that the occurrence of communicable diseases among school children may be minimized.

RESOLUTIONS ADOPTED AT THE TWELFTH ANNUAL CONVENTION OF THE AMERICAN SOCIETY OF CLINICAL PATHOLOGY*

As a definite program for our membership in its public relations we would recommend:

(1) The continued maintenance of the highest professional standards in our own work.

(2) The wise use of our influence to encourage legislation which will help preserve efficient laboratory service for the medical profession.

(3) Wider contacts between our members and other physicians to the end that they, as our associates, may know that as medical consultants and laboratory directors we are worthy of our hire.

(4) Active coöperation with all attempts to furnish more accurate tissue diagnosis.

(5) An official offer of coöperation by this Society in any organized plan for adjusting the economic side of medical care to the national budget.

RESOLUTIONS CONCERNING STATE LABORATORIES

Whereas, It is the recognized purpose of the Boards of Health of the various States to conserve the public health by measures for the prevention and control of communicable diseases, and

Whereas, Laboratories under the direction of many State Boards of Health have extended their activities beyond this field, for various reasons, and

Whereas, It is believed that the welfare of the public and of the medical profession will be best conserved if the activities of Public Health Laboratories are confined to measures for the prevention and regulation of communicable diseases, and to laboratory service for the indigent sick and for legal wards of the State,

Be It Resolved, That two propositions be recommended for the consideration of State Medical Societies.

1. That the furnishing of general diagnostic service by State Laboratories tends to prepare the public conscience for the acceptance of added varieties of State medical practice.

2. That while the cost of true public health laboratory service is a legitimate governmental expense, the extension of this service into the field of general laboratory diagnosis is an unnecessary and unwarranted burden upon the taxpayers of the State.

These two points for criticism in the State Laboratory situation are very apparent in clinical pathologists and are worthy of consideration by the entire medical profession. It is unwise to ask the State to practice medicine in the laboratory unless we are willing for it to spend still more public money and practice medicine in the ward and in the operating room.

The State Medical Societies can, and we believe they should, exert a powerful influence in keeping the demands made upon State Laboratories within the limits of truly public health functions. The Societies also can encourage the State Boards of Health to keep general diagnostic work out of their laboratories by regulation, and to inquire that each specimen from an indigent patient be accompanied by a card signed by both physician and patient. On this card the patient shall state: "I declare on my honor that I am financially unable to pay for this laboratory test and I know that the State Labora-

*Part of the report of the Committee on Public Relations of the American Society of Clinical Pathology as it appeared in the *American Journal of Clinical Pathology*, Vol. 3, No. 5, September, 1933.

tory makes no charge for acid tests." The physician's statement shall read: "I state, on my honor, that this patient is financially unable to pay, and that I am making no charge for my services to this patient."

C. W. MAYNARD, *Chairman.*

Motion made and seconded for acceptance of report. Carried.

It was further moved and seconded that a copy of this report be sent to each Counsellor of the Society, the American Medical Association, the American Surgeons, American College of Physicians, Secretaries of the State Boards of Health and Directors of the State Boards of Health. Carried.

Moved and seconded that a letter be sent to the Presidents and Secretaries of the State Boards of Health who have contributed by their recent actions in the movement to withdraw services in the general field of clinical pathology and have attempted to restrict their activities to strictly public health problems. Carried.

RECENT DEATHS

CHURCH — LUCY BARNEY HALL CHURCH, M.D., a retired physician of Dedham, Massachusetts, died December 28, 1933. She was born in 1866 and graduated from the Boston University School of Medicine in 1893.

She was active during the World War as a lecturer on social hygiene. She is survived by E. Norman Church, husband by her second marriage. A son by her first marriage, Fred Cahill Hall, served overseas.

POLLOCK — JACOB T. POLLOCK, M.D., of Chelsea, Massachusetts, died December 28, 1933. He was born in Russia in 1889. He graduated from the College of Physicians and Surgeons of Boston in 1913.

He is survived by a brother, Samuel Pollock, and three sisters, Mrs. Sarah Matzkin, Mrs. Dora Batzkoff and Mrs. Beatrice Gesson.

MAYNE — JAMES ELMO MAYNE, M.D., died at the Winchester (Massachusetts) Hospital December 25, 1933, of pneumonia.

Dr. Mayne was born in Drayton, Ontario, in 1901, the son of James and Sophronia Mayne. He graduated in medicine from the University of Toronto Faculty of Medicine in 1926 and joined the Massachusettts Medical Society in 1932.

He was also a Fellow of the American Medical Association. He served as intern at St. Michael's Hospital, Toronto, and the Williamsport Hospital, Pa., and later on the staff of the McLean Hospital in Waverley, and the Ring Sanatorium and Hospital in Arlington.

Previous to his death, he was practicing in Winchester. He is survived by his mother and two brothers. Interment was at St. Martin's cemetery, Drayton, Ontario.

HILLS — WILLIAM BARKER HILLS, M.D., of Montclair, New Jersey, died December 24, 1933, at the age of 83 years. He graduated from the Harvard Medical School in 1874, and was associate professor of Chemistry at the Harvard Medical School for many years up to 1904. The title of professor emeritus was later conferred upon him.

After leaving the medical school, he had charge of the chemical laboratories of the New England Mutual Life Insurance Company of Boston, until he retired in 1927. He was a member of the Massachusetts Medical Society from 1874 until he retired in 1915. He is survived by his widow, Miss Carrie M. (Sleeper) Hills, and a daughter.

SHURTLEFF — WALTER D. SHURTLEFF, M.D., of Plymouth, Massachusetts, died in that town at the Jordan Hospital, December 24, 1933.

He was born in Washington, D. C., and received his M.D. degree from Howard University School of Medicine of Washington.

Before moving to Plymouth, he practiced in Kingston. He joined the Massachusetts Medical Society in 1899. His membership was terminated in 1910.

He is survived by his widow, a daughter, Miss Flora Shurtleff, two sons, Albert H. Shurtleff of Plymouth, and F. Lebaron Shurtleff of New York.

NOTICES

COURSE ON MEDICAL BIBLIOGRAPHY

BOSTON MEDICAL LIBRARY
1934

January 9: Introduction. Dr. C. F. Painter.

January 19: Sources of Information. James F. Ballard.

January 23: Sources of Information. James F. Ballard.

January 30: How to Go about the Investigation of a Medical Subject. Dr. W. C. Quinby.

February 6: The Making of Medical Books. Dr. Henry Viets.

February 15: Incunabula. James F. Ballard.

Tuesdays at 8 P.M. except for the second meeting which will be on Friday, January 19.

Boston Medical Library,
8 The Fenway.

ANNOUNCEMENTS

HELEN SINCLAIR PITTMAN, M.D., announces the removal of her office to 412 Beacon Street, Boston.

FRED FINKLE, M.D., announces the removal of his office to 37 Columbia Road, Dorchester, Mass.

ROY E. MABREY, M.D., announces his association with E. A. Codman, M.D. at 227 Beacon Street, Boston.

REPORTS AND NOTICES OF MEETINGS

THE PHYSICIANS ALLIANCE

The regular meeting of the Physicians Alliance took place on Thursday, October 26, 1933, at 8:30 P.M. at the Whittier Street Health Unit, Roxbury.

Dr. Henry M. Landesman, Chairman, discussed the purposes and plans of the Physicians Alliance. He stated that this society was interested only in the economic welfare of the physicians and that it intended to coöperate in every way with the local district, state, and national organizations. He felt that certain things should be worked out and presented to the other organizations so that speedy action could be attained.

"The economic conditions of the physician have been undergoing a change to the detriment of the physician for several years. The depression naturally made things worse but the fundamental trouble was not improving but getting still worse; that the public, due to the tolerance, genuine kindness, honest philanthropy, and poor business ability of the physician, were taking advantage of and abusing the generosity and the humanitarian principles of the physician and were getting something for nothing. If the physicians were earning a living they would still continue as heretofore, but now, since a living is being denied them, they are forced with their backs to the wall to come out to battle for fair play. When among these fine men and women some are forced by exigencies and circumstances to apply for aid to prevent the possibility of being put out of their offices and homes, and to prevent their families from starving, it is but natural and human for us to get together and obliterate the underlying causes of the situation.

"The physician is always ready to help the indigent and the poor, but those in comfortable circumstances must be made by explanation and even shame to pay. The physician, during the past decade, has made tremendous progress in teaching the public preventive medicine. Physicians now rarely see cases of typhoid, diphtheria, smallpox, malaria, dysentery, and other diseases which used to provide a great deal of practice, and yet the public is unappreciative of the elimination of the pain, suffering and cost. We ask for no favors, only for fair play. We must be able to meet our expenses.

"We, therefore, hope that by bringing the various abuses of the public to the physician and the hospital and of some hospitals to the physician to an honest discussion among ourselves, we will be able to eliminate those practices that are harmful.

"The guest speaker for this evening is a physician whom you all know, who has shown his mettle and ability in the various positions which he has held previously at Harvard Medical School and then at the Crile Clinic in Cleveland, Ohio, and who is now holding the foremost position in the country, successor to the world's greatest brain surgeon, Dr. Harvey Cushing, — the Moseley Professorship of Surgery at the Harvard Medical School, and Surgeon-in-Chief of Peter Bent Brigham Hospital, Dr. Elliott C. Cutler, who will read a paper on the 'Relation of the Physician to the Hospital Out-Patient Department.'"

THE RELATION OF THE PHYSICIAN TO THE HOSPITAL OUT-PATIENT DEPARTMENT*

BY ELLIOTT C. CUTLER, M.D.

Members of the Physicians' Alliance: I am very grateful to you for being allowed to meet you tonight. Perhaps the busy practitioner does not realize how much those people whose lives are confined largely to hospital practice delight to get out now and then and meet their colleagues under such circumstances.

Your problem, and the topic of this evening's meeting, "The Relation of the Physician to the Hospital Out-Patient Department," is part of the general economic situation of today. I personally do not believe that the present financial situation has brought anything new into medical practice. It is only that the pinch of our present depression has focussed our minds on the general trends of modern medical practice, trends which have year by year encroached upon the province of the general practitioner of medicine. State medicine, state sanatoria for tuberculosis and cancer in addition to increased public health measures, and the increasing tendency for all forms of health insurance in addition to accident cases, have gradually taken away from the regular practitioner a large part of his work. At the same time that this has been going on, nurses have been better educated and by being better able to help doctors have made it possible for fewer doctors to care for more people. And always we have continued to turn out in this country more doctors than the people need!

The financial depression of today has emphasized these changes and we now see clearly the results of what has really been going on for many years. To this extent the present financial difficulty is of benefit and should allow us to crystallize out of the welter of criticisms and suggestions what is best for the progress of modern medical practice. It is obvious that there will be changes. One of these must be the reduction in the number of new doctors, and could some of those who are urging larger classes in medical schools be here tonight, they would visualize a dilemma which their desires may well make worse.

The topic of tonight is really incomplete in that it should be the relation of hospitals to the practitioners of medicine, for there are two distinct phases in this possibility that hospitals may be taking practice away from the general practitioners. The first phase has to do with patients consulting hospital dispensaries who might possibly afford to go to a doctor, and the second phase has to do with

*Read before The Physicians' Alliance, Boston, October 26, 1933.

the hospitals continuing to carry patients, referred to them by a doctor, in their dispensaries rather than returning those patients to the physician. Before speaking tonight of the immediate situation with which I am familiar here in Boston, i.e., the relation of the Peter Bent Brigham Hospital to the practitioner, I wish to recall to you that I went through a much more serious economic difficulty in Cleveland where the depression has been far more deeply felt. The same situation there, only more intense and engendering a bitterness not seen here tonight, reached such a pitch that it was finally agreed that patients would not be admitted to hospital dispensaries except when referred by a doctor. This rule both protected the hospitals, which found they could not carry the load financially, and possibly eased somewhat the practitioners' situation. But many evils resulted from such an arbitrary ruling. It was soon found that doctors were accepting small payments just to give a patient a letter saying that he could go to a dispensary and receive free care! Moreover, certain patients preferred the hospital service and objected to any infringement of their rights to choose their own medical advice.

Now I should like to discuss the matter of this evening in relation to the Brigham Hospital. When the patient comes to the dispensary, every attempt is made to find out whether he has a physician. If we can determine who the physician is, we write to him at once and receive his opinion as to whether we should treat the patient or send the patient back to him. We also enquire at this time what it is he thinks should be investigated in his patient. If no doctor can be located, the social service department tries to determine the financial status of the individual. We have to accept the patient's word as to his income and job, for a more elaborate survey of every individual's financial status would be too great a burden for us. As a matter of interest, previous investigation has already determined that only about two per cent of people lie about this at the hospital admitting offices. Probably, therefore, it would not be financially worthwhile to carry investigations further. It is true that from time to time patients' statements as to their employment are checked up. When patients are found to have an income above the limit set by hospital administrators as suitable for dispensary care, they are refused treatment in the Peter Bent Brigham Hospital Out-Door Department.

The second phase of the problem has to do with the admission of patients to the hospital. On the medical service, when a patient is admitted, a card is sent to the referring doctor, asking him to come on ward rounds and see the patient with the hospital staff. On the surgical service, pre-operative contacts are attempted and always the recommending physician is notified of the day and time of the operation. Doctors who accept these invitations probably never lose their patients for a part of the loss of the practitioner's patients when his clientele enter a hospital is due to the fact that during this period of hospitalization he does not continue to take an active interest in his cases. Upon the discharge of patients from the hospital, a summary note of what happened to the patient is sent to the referring physician and we try to refer all cases back to the doctor who sent the case to the hospital. Unquestionably, slips occur in this custom, but they are not very frequent and we constantly check back into this matter. It is true that in surgery we run a follow-up clinic and ask patients to come back from time to time to check up on their end-results. But these follow-up examinations have nothing to do with the dispensary itself and are merely that we may determine the efficacy of the treatment we give. In order that cards to patients asking for follow-up examinations may not be misconstrued, we are now sending out notices to the patients' physicians whenever such an end-result examination is asked for so that they may fully appreciate that this examination is without fee and is not for therapy.

These are the protections with which we try to surround ourselves so that the practitioner may feel we are in the community to help him as best we may. You must remember that the people in charge of the large hospitals are vitally interested in your goodwill. They know that their hospital cannot be successful unless you have faith in it. We have, therefore, the most forceful reasons for doing everything we can to retain your good wishes.

With this statement it must appear to you that the hospital is not in any competition for practice with the general practitioner of medicine. Indeed it is really there for your benefit and I am certain that none of you would prefer to work in a community where there was not a good hospital. The hospitals may make mistakes and possibly some of these can be avoidable by modification of the system of admission in some hospitals, but if the outline which we follow at the Brigham Hospital is carried out as carefully as possible, there will be the minimum of such mistakes.

Dr. Irving J. Walker, Clinical Professor of Surgery, Harvard Medical School and Surgeon-in-Chief of the Fifth Surgical Service, Boston City Hospital, discussed the paper as follows:

I am very glad to be with you tonight to discuss this very interesting subject. It is quite natural for human nature to endeavor to get something for nothing, or for as little cost as possible. This applies to medicine as well as to other aspects of life.

While I am a member of the Staff of the Boston City Hospital, I do not, of course, represent the Staff or the Administration in my statements.

I quite agree with all that has been said regarding the abuse of the charitable hospitals in and about Boston by those people able to pay for medical services. It may be possible that the Boston City Hospital is one of the chief offenders in this abuse. At times I am personally rather upset when I see

these patients that we have operated upon without any charge in our hospital wards, having special nurses or perhaps occupying private rooms.

To correct this abuse in hospitals incorporated under private ownership is one problem and to correct it in a municipal institution like the Boston City Hospital is quite another matter, especially where the political aspect of the situation is a factor. I have no suggestion to make that would absolutely solve the problem, except that I feel sure that, if this body or its representatives desire to consult with the Trustees of the Boston City Hospital, those gentlemen would be glad to talk the matter over and offer whatever help they could render in the solution of the problem.

Dr. Hilbert F. Day, Clinical Professor of Surgery at Tufts Medical School and Surgeon-in-Chief, Boston Dispensary, said that as it had not been stated whether by physician you meant those working in the clinics or those working outside, he would begin his remarks by saying that he felt some method should be worked out whereby insurance cases, automobile accidents, etc., appearing for treatment in Out-Patient Departments and whose illnesses were being paid for by insurance, should mean some compensation to the physicians treating them. He said that this could only be accomplished by concerted unified action by the physicians of the state.

In regard to physicians outside of the hospital, he spoke of the present trend in hospitals toward coöperation with the practising physicians. He felt that the facilities for diagnosis which are so abundant in hospitals should be made more accessible to the practising physicians and that opportunities for observing work in clinics should be given them.

He felt that fewer patients would be a loss to hospitals if the doctors maintained an interest in them and went to see them when they were sick.

He stated that the hospitals had no need to build up their clinics and suggested that it might be advisable for a physician to sit in with the admitting social service representative on admissions to pass on the medical necessity or emergencies as well as on the financial status. He said that the principle of consulting the private physician who had seen the patient some time within the last few years should be more general, and that this physician should be requested to state whether he thought the patient should come to a clinic or whether he, the physician, would prefer to treat the patient during his financial embarrassment.

He concluded by saying that he felt that there should be at least a regular yearly review of patients going to clinics, for the fact that today a patient is deserving of charity care in a hospital might not be true a year from now and that the principle which is prevalent in Boston hospitals now, ONCE IN ALWAYS IN, should not be maintained. At present if a patient is once admitted to an out-patient clinic and gets a card, he can return to that clinic two months or three years afterward and not be requestioned about his financial situation.

Dr. Day expressed the opinion that the free discussions of this problem by a group such as the Physicians' Alliance multiplied by similar organizations throughout the State would inevitably tend toward better conditions in the practice of medicine, at least from the practitioner's standpoint.

Dr. I. A. Shain:

Mr. Chairman and Gentlemen:

The serious condition confronting the medical men at the present day may be ascribed to three causes.

1. The acute depression that has been facing the country for the past four years.
2. The extensive specialization in medicine.
3. The social service problem.

The first, the economic problem, need not be discussed here.

The second is the extensive specialization in medicine. The practice of medicine has become so broad that no single medical practitioner is able to treat every case that comes before him. There are times when he requires the aid of specialists.

In times of prosperity the patients were anxious to be referred by their doctor to these specialists when a question of diagnosis came up. However, as the economic condition became more acute, these patients found it difficult to pay the family doctor, let alone the various specialists. As a result of this, they flocked to the hospitals, where they were welcomed and received care that the specialists provided.

Therefore, I agree with what Dr. Hilbert F. Day has so ably stated; that the general practitioner should have greater access to the hospitals; so that a patient who is unable to pay and requires special examinations can be referred to the hospital for that particular special examination. For example, a patient requiring a gastro-intestinal x-ray examination or a special examination of the heart including electrocardiograph or other special work could be referred to the x-ray department or to the cardiac clinic of the hospital for consultation. After the examination the patient could be sent back to the doctor with a clinical report and diagnosis by a member of the staff of the hospital. In this manner the patient may receive the best attention and still be under his physician's care. Furthermore, no patient should be admitted to the hospital without a letter from his physician. This would be no hardship to the patient, because every individual has a family physician who knows his financial standing and could determine whether that patient is a suitable case for a public or semi-public hospital. The doctor should be welcome to participate in the visits and discuss the patient's condition. Upon the discharge of the patient, he should be immediately referred to the doctor and not to the Out-Patient Department. Such a procedure would eliminate hospitalization of the patients who can well afford private care and bring

greater coöperation between the doctor and the hospital.

The third is the social problem. There are in this community so-called social service and welfare workers who make a house-to-house canvass, examine the members of the family, and take it upon themselves to make a diagnosis and insist on taking these individuals to various hospitals for treatments and operations; many times against the family's wishes. They even come with automobiles and transport them to and from these hospitals. This, I consider a very serious problem. In many instances the family could well afford private care and also in many cases the members of the family are averse to such high-handed methods. I feel that the family physician is well able to determine what treatments and what operations are really necessary.

In the discussion it was pretty well agreed by all that there was something fundamentally wrong with conditions as they are and that hospitals cannot get along without physicians and that physicians cannot get along without the hospitals; that a great many patients have acquired the habit of getting something for nothing; that the policemen and firemen and city employees should not be allowed free services at the hospitals; and that, if conditions are to be improved and changed in any way, it would have to be done by a concentrated effort on the part of the physicians themselves.

The following physicians also took part in the discussion: Dr. D. L. Lionberger, Dr. Max Ritvo, Dr. John F. Casey, Dr. Morris N. Davidow, Dr. John A. Hall, Dr. A. A. Levi, Dr. William Thau, and Dr. Louis Feldman.

BOSTON CITY HOSPITAL

Staff Clinical Meeting

The Staff Clinical Meeting of the Boston City Hospital took place on Wednesday, December 6, at 8:15 P.M., in the Thorndike Amphitheater. It was given over to a consideration of genito-urinary problems. Dr. Joseph H. Burnett acted as chairman. The program read as follows:

Nephrectomy in Renal Tuberculosis and the Value of Long Continued After-Care, by Dr. Herbert H. Howard. Discussion by Dr. George Gilbert Smith.

Nephropexy for Ureteral Kinks. A Review and Results of Ninety Cases, by Dr. Augustus Riley. Discussion by Dr. J. Dellinger Barney.

Perineal Injection of Mercurochrome in Prostatic and Vesicular Infections, by Dr. Gordon D. Atkinson.

Treatment of Acute Gonorrheal Arthritis of Knee, by Dr. William F. Cotting. Discussion by Drs. H. H. Howard, A. Riley, and O. J. Hermann.

In the first paper of the evening, Dr. Howard recommended nephrectomy in a bilateral tubercular process if the remaining kidney showed clear urine and a normal pyelogram. Operation, however, was contraindicated in those patients who showed definite ulcerative lesions demonstrable by pyelography. He further recognized the existence of cases of healed renal tuberculosis. In these latter, very much, he thought, depended on good and prolonged adequate after-care; this should include routine examination of the urine with one or more guinea-pig tests. The urine for the guinea-pig test should be a concentrated catheter specimen.

In his operations, Dr. Howard found that removal with the diseased kidney of that portion of the renal fat adherent to the tubercles, would cut down the number of resulting renal sinuses; moreover, that Conroy's Solution successfully cleared up two of his cases of persisting renal sinuses that followed nephrectomy. He further advocated ureterectomy in tuberculous ureters showing a good deal of beading.

In discussing the paper Dr. Smith, unlike Dr. Howard, felt that ether was not objectionable as an anesthetic, and that he did not hesitate to use it in cases without pulmonary tuberculosis. He is more afraid of gas-oxygen inhalation. Moreover, in the management of bilateral renal tuberculosis, he indicated that providing one kidney was entirely destroyed and the patient not in extremis, it was perfectly proper to remove the diseased one. He stressed the fact, however, that since it usually takes ten years, approximately, for an early infected kidney to become useless, it is unwise to remove either kidney if there is a fair degree of function in each of them. In addition, one should not be in a hurry to operate, but should provide the patient with absolute rest and sanatorium care for a period of several months prior to an operation; in this way, giving the patient a chance to build up a resistance to his disease, and protecting him against the dissemination of the tubercular organisms incident to the operation.

Dr. Augustus Riley concluded from an analysis of ninety cases, that nephropexy for ureteral kinks was still worth while if properly done. Some of his patients, following such operations, have successfully gone through pregnancy with the kidneys remaining normal throughout the entire period. Moreover, he observed that any previous hydronephrosis, incidental to the kinking, soon cleared up. All of his cases showed pain as the chief symptom; this was like that of the typical renal colic. Nephropexy relieved the colic in all instances. However, nearly all of the patients had a dull dragging pain following the operation, which lasted for some time, till the kidney got used to its new position.

The causes for the kinking in the ureters was found to be due to one of the following conditions: an aberrant artery, retroperitoneal metastatic malignant disease, or tough fibrous bands. Females have kinked ureters, Dr. Riley found, twice as often as males, with the kinking usually taking place twice as often on the right side as on the left.

The third speaker, Dr. Gordon Atkinson, described the treatment of prostatic and vesicular infections by the perineal injection of mercurochrome into

these organs. The method was utilized in twenty-five cases that had not responded well to the ordinary routine manner of treatment with sounds, silver nitrate irrigations, etc. An injection consisted of five cubic centimeters of a three per cent mercurochrome solution. Following several injections, nineteen of the twenty-five refractory patients showed definite progress. In all of the cases, the patients were relieved of pain. Six cases, however, did not show improvement of the main lesions. In none of these patients were there any bad results, severe reactions, or abscess development, as the result of the injections. Dr. Atkinson does not believe that this method, in itself, is sufficient for the successful treatment of chronic prostatitis.

Dr. William F. Cotting presented the results of the treatment of "Acute Gonorrheal Arthritis of the Knee," by the incision and irrigation of the joint with 1 to 20,000 bichloride of mercury solution. Twenty cases had been given the treatment, with good results. In four of these, positive tests for the gonococcus were obtained with material from the infected knee joints. In all the cases spinal anesthesia worked well. Dr. Cotting brought out that the insufflation of air into the joint, as a method of treatment in acute and subacute cases, was given up after the occurrence of a fatality in one of the ten patients so treated; the consensus in this instance indicated death as due to an air embolus.

WILLIAM HARVEY SOCIETY

The William Harvey Society of the Tufts College Medical School held its December meeting in the amphitheatre of the Beth Israel Hospital on Friday evening, December 8, 1933. Dr. Harrison S. Martland, Pathologist to the Newark City Hospital and Medical Examiner for Essex County, New Jersey, was the principal speaker. Dr. Martland is well known especially for his recent thorough investigation of the radium dial-painter poisonings in New Jersey. The speaker was introduced by Dr. Timothy Leary, Medical Examiner for Suffolk County, Southern District, after the latter had briefly reviewed the history of the medical examiner system as developed in Massachusetts from 1877. The subject of the evening was "The Causes of Sudden Death."

The speaker began by defining what he meant by sudden death. Homicides, suicides, violent accidents, death suddenly when in apparent health, death in the absence of a physician or within twenty-four hours of hospital admission, and deaths under suspicious or unusual circumstances come under the category of "sudden death". The function of the Medical Examiner is the investigation of deaths due to crime or negligence, as well as the investigation of sudden and natural deaths under unusual circumstances but where there is no direct evidence of violence.

Sudden deaths due to crime and negligence were briefly passed over with some very vivid accounts of life ending in this manner, and then, with the aid of lantern slides, Dr. Martland proceeded to review 1000 autopsies on cases of sudden death from natural causes, limited to victims over ten years of age, with the quotation of statistics under each heading. Six hundred and ninety-seven persons out of the thousand, or 69.7 per cent, died of diseases of the heart and blood vessels. Most of these deaths were due to the various manifestations of so-called "arteriosclerotic heart disease," hypertensive heart disease and diseases of the coronary arteries. The pathological and clinical sequences in coronary thrombosis were depicted on the screen: the well-known progression from thrombosis to infarction, to necrosis or healing and repair, with early pericarditis or in some instances mural thrombus formation. The descending branch of the left coronary artery is the most common site of acute coronary thrombosis. Cardiovascular syphilis was found to be the cause of 14.4 per cent of the deaths in the series, or 20.8 per cent of diseases of the heart and blood vessels. The course of this disease in the production of pathological changes in the aorta was briefly reviewed. Making up the 14.4 per cent total were aortic regurgitation, 4.2 per cent (death due to acute dilatation or an anginoid type of failure), coronary stenosis, 4.2 per cent, aneurysm 5.5 per cent, and 0.5 per cent due to unusual syphilitic conditions. Patients lived longer with an aneurysm than with any other kind of cardiovascular lues. A high diastolic pressure in the presence of competent aortic valves causes a "blow-out" in the weakened aortic wall and death finally ensues from sudden rupture. Rheumatic heart disease accounted for 93 cases, 9.3 per cent, in the series, or 13.3 per cent of the deaths due to diseases of the heart and blood vessels. In these 93 cases, 64.4 per cent of deaths were due to valvular lesions and 15 per cent to myocardial lesions. The remainder were caused by subacute bacterial endocarditis (four cases) and aortic stenosis (12 cases). Spontaneous rupture of the aorta (non-syphilitic) occurred in 17 cases of the thousand, congenital heart disease in one, and miscellaneous diseases of the heart and blood vessels (tumors, etc.) in five.

Diseases of the nervous system accounted for 103 cases, 10.3 per cent. Cerebral hemorrhage was found in 34, spontaneous subarachnoid hemorrhage in 12, brain tumor in 10, brain abscess in one, alcohol poisoning in 30, and status epilepticus in seven. The remainder were due to miscellaneous causes.

Diseases of the respiratory system could be held accountable for 115 of these sudden deaths, 11.5 per cent of the total. The most common causes in this group were lobar pneumonia and acute edema of the larynx. Acute hemorrhage caused 17 deaths, embolism two, and so-called "status lymphaticus" 12. The hypothetical nature of this last entity in the light of recent opinion was questioned by Dr. Martland, who believes that in these few cases of sudden death generalized lymphoid hyperplasia

(elsewhere in the body as well as in the thymus) cannot be overlooked and passed by as insignificant.

Dr. Leary briefly compared his investigations with those of Dr. Martland and adjourned the meeting shortly after ten o'clock.

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society of the Tufts College Medical School, will be held at 8:00 P.M. on January 12, in the auditorium of the Beth Israel Hospital, Boston.

The speaker will be Dr. William Allen Pusey, formerly President of the American Medical Association. Subject: "The History of Syphilis." Chairman: Dr. William P. Boardman, Professor of Dermatology, Tufts College Medical School.

Members of the medical profession are cordially invited to attend the meeting.

HOUSE OFFICERS' ASSOCIATION OF THE BOSTON CITY HOSPITAL

MONDAY, JANUARY 8, 1934

Subject: "The Acute Abdomen."

8:00 P.M. Presentation of Cases.

8:15 P.M. Dr. William R. Morrison. "Acute Perforative Lesions of the Upper Abdomen."

8:35 P.M. Dr. Elliott C. Cutler. "Non-Perforative Abdominal Trauma."

8:55 P.M. Dr. Franklin W. White. "Medical Aspects."

9:15 P.M. Dr. Irving J. Walker.

Discussion.

The meeting will be held in the Thorndike Amphitheatre.

ROBERT T. PHILLIPS, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, January 16, at 8:15 o'clock.

PROGRAM

Presentation of Cases.

"A Trip to the East Coast of Greenland." By Dr. W. G. Smillie with motion pictures.

JOHN HOMANS, M.D., *Secretary.*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, January 16, 1934, at 12 noon. The speaker will be Maurice Fremont-Smith, M.D., Assistant Physician to Massachusetts General Hospital, and his subject will be "The Treatment of the Milder Neuroses." All physicians are cordially invited to attend. The usual luncheon will be served at 1 o'clock.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

On Tuesday, February 20, at 5 P.M. at the Metropolitan State Hospital, Waltham, there will be a meeting of the Middlesex South District Medical Society.

Speakers:

Dr. Roy D. Halloran — "The Metropolitan State Hospital—Purpose and Development."

Dr. Malcolm J. Farrell — "Demonstration of Two Similar Psychotic Entities, Postencephalitis Lethargica and Dementia Praecox, Catatonic Type."

Dr. Philip F. Hilton—"Demonstration of Injection Treatment of Varicose Veins in a Mental Hospital Population."

Dr. Clifford D. Moore—"Mental Disease Occurring in the Course of Physical Disease, with Particular Reference to Pulmonary Tuberculosis."

Buffet luncheon after the meeting.

ALEXANDER A. LEVI, M.D., *Secretary.*

MASSACHUSETTS GENERAL HOSPITAL

A clinical meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building on Thursday, January 11, at 8:15 P.M.

PROGRAM

X-ray Diagnosis of the Esophageal Varices. By Dr. Richard Schatki.

Clinical Significance of Bleeding from Upper Intestinal Tract. By Dr. Chester M. Jones.

Massive Hemorrhage from Duodenal Ulcer. Surgical Treatment. By Dr. Arthur W. Allen.

Discussion.

Physicians, medical students, nurses and social workers are cordially invited.

COMMITTEE ON HOSPITAL MEETINGS.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

January 4—Faulkner Hospital Clinical Meeting will be held at 5:00 P.M.

January 8—House Officers' Association of the Boston City Hospital. Subject: "The Acute Abdomen". See notice elsewhere on this page.

January 9 - February 15—Course on Medical Bibliography, Boston Medical Library. See page 50.

January 11—Massachusetts General Hospital. See notice above.

January 12—William Harvey Society. See notice elsewhere on this page.

January 15, 1934—Boston Medical History Club. Speaker: Professor Henry E. Sigerist, Director of the Institute of Medical History of Johns Hopkins University.
JAMES F. BALLARD, Secretary.
Boston Medical Library, 8 The Fenway, Boston.

January 15, 16, and 17, 1934—American Conference on Birth Control will be held at the Mayflower Hotel, Washington, D. C.

January 16—Harvard Medical Society. See notice elsewhere on this page.

January 16—South End Medical Club. See page 56.

February 7—Annual dinner of the Tufts College Medical School Alumni Association at the Boston City Club, 6:30 P.M.

February 12—House Officers' Association of the Boston City Hospital. Subject: "Forensic Psychiatry". Speakers: Dr. A. Warren Stearns and Dr. Abraham Myerson. See page 56.

February 16 and 17, 1934—The New England Hospital Association is holding its Twelfth Annual Meeting at the University Club, Boston. For details write Dr. A. G. Engelbach, Massachusetts General Hospital, Boston.

April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schniz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

September 4, 5, 6, 1934—International Union Against Tuberculosis will be held in Warsaw. For particulars address The National Tuberculosis Association, 450 Seventh Avenue, New York, N. Y.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

February 7—Council Meeting, Boston.

Wednesday, March 7—Lynn Hospital. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Frank H. Lahey, Boston. Subject to be announced. Film Electrocardiogram.

Wednesday, April 4—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speakers: Dr. Elliott P. Joslin and Dr. Howard F. Root, Boston. Subject: "Tuberculosis Complicating Diabetes."

Thursday, May 3—Censors' Meeting, at Salem Hospital, 3:30 P.M.

Tuesday, May 8—Annual Meeting. Salem Country Club, Forrest Street, Peabody. Dinner at 7. Speaker to be announced. Subject to be announced.

RALPH E. STONE, M.D., Secretary.
221 Cabot Street, Beverly, Mass.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.
Sunderland, Mass.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Meetings will take place on January 10 at Melrose, March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, M.D., Secretary.
76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on January 31, and April 25 (Annual Meeting).

T. A. STAMAS, M.D., Secretary.
226 Central Street, Lowell, Mass.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

February 20—See page 56.

NORFOLK DISTRICT MEDICAL SOCIETY

Postgraduate Course. See page 1184, issue of December 7.

January 28—Tentatively Roxbury Masonic Temple, 8 P.M. Continuance of Symposium on Diseases of the Blood. Dr. Dameshek and Dr. Straus.

February 27—Place, time, subject, and speaker to be announced.

March 27—Place and time to be announced. Dr. Edward A. Edwards and Dr. Henry H. Faxon. Symposium on the Treatment of Varicose Veins.

May—Annual Meeting. Place, time and program to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline, Mass.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

January 4—12 noon at Norfolk County Hospital.

February 1—12 noon at Norfolk County Hospital. Stated Meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

N. R. PILLSBURY, M.D., Secretary.
Norfolk County Hospital, South Braintree, Mass.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 31—General Meeting in association with the Boston Medical Library and the Trudeau Society. Speaker: Dr. Lawrason Brown of Saranac Lake.

March 28—Clinical Meeting at the Massachusetts Memorial Hospitals.

April 25—Annual Meeting at the Boston Medical Library. Election of Officers. Scientific Program, titles and speakers to be announced.

The Medical Profession is cordially invited to attend all of these meetings.

JAMES H. MEANS, M.D., Vice-President.
GEORGE P. REYNOLDS, M.D., Secretary,
311 Beacon Street, Boston, Mass.
ALEXANDER S. BEGG, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

All meetings to be held on Wednesdays as follows:

January 10—Dinner and scientific program at the Worcester City Hospital, Worcester, Mass.

February 14—Dinner and scientific program at the Worcester State Hospital, Worcester, Mass.

March 14—Dinner and scientific program at the Memorial Hospital, Worcester, Mass.

April 11—Open date.

May 9—Annual Meeting. Time and place to be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester, Mass.

BOOK REVIEWS

Obstetrics and Gynecology. Volume II. By 80 Leading Specialists. Edited by Arthur Hale Curtis, M.D. Complete in 3 Volumes and Separate Desk Index. 3500 pages, 1664 illustrations, many in colors. Philadelphia and London: W. B. Saunders Company, 1933. Price per set, cloth $35.00 net.

The second volume of Obstetrics and Gynecology of which Dr. Curtis is the editor begins with "Pathology of Labor," Section VIII. The authors take up in order dystocia due to contracted pelvis, to abnormalities of the genital tract, and anomalies of the expulsive forces, three very satisfactory chapters. The next chapter is on "Anomalies of the Passenger," and Piper of Philadelphia writes a most stimulating article. He has somewhat encroached upon the subjects of other writers, and some of the advice he gives is entirely contrary to that of these authors. The result may be very disconcerting to the student.

The chapter on "Hemorrhage" is the poorest in the book. It contains many statements which are not in line with the best obstetric thought of the day, the illustrations are poorly chosen, there is not one original illustration in the article, and it contains no bibliography. It should be rewritten.

The section ends with a short chapter by Irving on "Prolonged Pregnancy" in which he takes a most satisfactorily conservative stand, in direct opposition to statements on the same subject in other parts of the book.

Section IX is headed "Pathology of the Puerperium," and Watson of New York has written a splendid article on "Puerperal Infection and Thrombophlebitis." It is in every way a most commendable chapter.

Section X is on "Operative Obstetrics." The various subjects are on the whole very well covered. There are remarks here and there to which it is natural that all cannot subscribe. It would seem unnecessary to devote as much space as Danforth has to Hebosteotomy, for at the present time it has no place in obstetrical procedures.

The illustration on page 329 showing a patient properly draped for forceps delivery seems to the reviewer to approach the absurd, for the technique cannot be carried out in a large majority of cases, it is totally unnecessary, and the reviewer does not believe that the results following such technique will be any better than if a more simple technique were advised. It is to be regretted that in none of these chapters on the technique of operative obstetrics is the preparation of the patient stressed or reference made to the previous description of the preparation of patients in the first volume.

Stander's article on "Caesarean Section" is outstanding. It is a sane, well-written article, with which every student of obstetrics should familiarize himself. Tucked in at the end of this section is an interesting though brief outline of the history of American gynecology by Kelly.

Section XI is on "Infectious Processes." Curtis writes on gonorrhea very satisfactorily, and Gellhorn's article on syphilis is thorough, clear, inclusive, and is followed by an excellent bibliography.

Section XII covers "Tumors of the Uterus," and Graves's article on fibroids is excellent in every way. Martzloff has a well-written and authoritative article on "Carcinoma of the Cervix," and Healy has one on "Carcinoma of the Body of the Uterus." Schumann has written an interesting chapter on "Other Tumors of the Uterus" covering the more rare types that occur.

In Section XIII tumors of the ovary and fallopian tubes are discussed. The volume ends with an adequate index.

On the whole it appears to the reviewer that the correlation in this volume is not so good as in the first. In a book that purports to be outstanding and authoritative, it would seem reasonable that more of the illustrations should be original than is the case. Many of them have been taken from previously published books, some of which are good, others of which should not have been reproduced. The reproduction of an illustration showing manual dilatation of the vulva with the patient unshaved is inexcusable in these days. Apparently the writer thought so well of it that he reproduced it a second time. Such illustrations will unquestionably give the student a wrong idea of what the proper technique in obstetrics should be. Except for the several outstanding chapters which have been spoken of, the book is a disappointment compared with what the reviewer expected from the first volume.

Migraine. Diagnosis and Treatment. By RAY M. BALYEAT, M.A., M.D., F.A.C.P. Associate Professor and Lecturer on Diseases Due to Allergy, University of Oklahoma Medical School. J. B. Lippincott Company, Philadelphia. 242 Pages. Price $3.00.

Dr. Balyeat has assembled in this volume all available data from history and literature, and reports the results of his personal study of approximately 350 cases in which a diagnosis of migraine was made. While other possible factors are given due consideration and a judicial and conservative tone is maintained, the work is mainly an exposition of the importance of allergy in the etiology, and of the practical value of treatment by elimination from the diet of foods to which the patient is sensitive. The diagnosis of true migraine is discussed and the treatment by measures other than control of the diet. Fifty-three illustrative cases of allergic headache are reported in detail.

BOOKS RECEIVED FOR REVIEW

Sex Habits, A Vital Factor in Well-Being by A. Buschke, and F. Jacobsohn. Published by Emerson Books, Inc. 204 Pages. Price $2.50.

The Pregnant Woman by Porter Brown. Published by Eugenics Publishing Company, Inc. 174 Pages. Price $2.00.

The Foundations of Nutrition by Mary Swartz Rose. Revised Edition. Published by The Macmillan Company. 630 Pages. Price $3.00.

Food Products by Henry C. Sherman. Third Edition. Published by The Macmillan Company. 674 Pages. Price $3.00.

Benign Tumors in the Third Ventricle of the Brain: Diagnosis and Treatment by Walter E. Dandy. Published by Charles C. Thomas. 171 Pages. Price $5.00.

The Surgical Clinics of North America. Volume 13—Number 6. Pacific Coast Surgical Association Number. Index Number. Published by W. B. Saunders Company. Pages 1277-1559. Per clinic year (February 1933 to December 1933). Price, Paper $12.00; Cloth $16.00 net.

Physiopathologie des Syndromes Endocriniens by Noël Fiessinger. Published by Masson et Cie. 312 Pages. Price 40 fr.

The Modern Treatment of Syphilis by Joseph Earle Moore. Published by Charles C. Thomas. 535 Pages. Price $5.00.

The Physician's Art by Alexander George Gibson. Published by The Clarendon Press at Oxford. 237 Pages. Price $3.00.

Traité de Physiologie Normale et Pathologique par M. M. L. Ambard, M. Arthus, E. Bachrach, A. Blanchetière, H. Cardot, A. Chevallier, A. Lacassagne, A. Policard, G.-H. Roger, J. Verne, F. Vlès. Publié sous la direction du Pr. G.-H. Roger et Du Pr. Léon Binet. Masson et Cie, Éditeurs. 1140 Pages. Broché: 145 fr. Relié: 165 fr.